TEXTBOOK OF BRITISH SURGERY

Edited by

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VOLUME ONE : THE ABDOMEN



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EDITOR'S PREFACE

THE advances of Surgery in the last twenty years have been so great that no one individual can master all the fields which they have opened. On the other hand it is important that candidates for the higher examinations should be familiar with the whole subject, for only thus will they be able to select for their future career the branch for which they are best adapted. This new *Textbook of British Surgery* aims to meet their requirements.

It has been compiled by some forty authors, each an acknowledged master in his own particular branch, and the aim has been to give a clear and succinct but complete account of the present position in each field. So rapid has progress in surgery been that several of these articles have been completely rewritten while the book was being compiled. At the moment they present an accurate view of surgical practice today on its highest plane. We hope that they will be of material use to the student in acquiring the knowledge which is necessary for his work, and that later on they may inspire him to add to that knowledge and by his own labours to develop still further the great subject to which he is devoting his life.

H. S.

London, 1955

GENERAL PREFACE

THIS work will be issued in three volumes, of which this one, the Surgery of the Abdomen, is the first. Each volume is complete in itself. The three volumes are planned to cover the entire field of surgery: diagnosis, pathology, prognosis and treatment. Sir Henry Souttar, as general editor, has collected around him a team of some 40 contributors, each a recognized specialist in his own subject. The standard aimed at is as complete an account as possible of the present position in each field of surgery as seen by the expert to whom the section has been entrusted. The names of the contributors are a sufficient guarantee that this standard will be attained. They are drawn from the leading medical schools and hospitals throughout the kingdom and represent the consensus of opinion in present-day British Surgery.

The volumes will be essentially clinical and practical, with such pathology as is necessary for diagnosis and treatment. Surgical procedures will be described, and the authors will discuss the advantages and disadvantages of each procedure in vogue, indicating their reasons for preferring one to another.

Each volume will be illustrated by original drawings, photographs, X-rays, diagrams and coloured illustrations. The audiences they are intended to interest are general surgeons, registrars, post-graduate students and those reading for the Fellowship and other higher examinations.

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CONTENTS

SURGICAL ANATOMY

GUY BLACKBURN

The anterolateral musculature of the abdominal wall, consisting of the Rectus and Pyramidalis, the External and Internal Oblique muscles and the Transversalis, is relatively constant in its arrangement. Weakening of the musculature with advancing age, variation in the amount of subcutaneous fat, visceroptosis and repeated operations or pregnancies may, however, alter the configuration to an appreciable extent. These factors have, therefore, to be taken into account in planning abdominal incisions.

The bony prominences of importance are:

(1) The ensiform cartilage, which is easily palpable and which lies opposite the lower border of the tenth thoracic vertebra.

(2) The costal margin, consisting of the fused seventh, eighth, ninth, and tenth costal cartilages.

(3) The pubic tubercle, which is much easier to find than the symphysis.

(4) The anterior superior iliac spine.

The eleventh and twelfth costal cartilages are unimportant in relation to abdominal incisions, except in the drainage of a subphrenic abscess or the Bernard Fey approach to the kidney. Here, the twelfth or eleventh rib respectively may be resected and its length may be best deduced from an X-ray. The last rib, in fact, often does not project beyond the margin of the Erector Spinæ muscle.

The *umbilicus* varies somewhat in position but is usually at the level of the lower border of the third lumbar vertebra. A line from the anterior superior spine through it reaches the opposite costal margin at the ninth costal cartilage. This may be employed as the surface marking of the fundus of the gall bladder.

The *Rectus* muscle is the most important single component of the abdominal wall in relation to laparotomy and a thorough understanding of its sheath is most important. This is formed by the division of the aponeurosis of the internal oblique muscle into two layers, the anterior blending with the external oblique aponeurosis and the posterior with that of the transversalis. In the lower third of the muscle, however, at a point mid-way between the umbilicus and the symphysis, this splitting ceases to occur, at the arcuate line, and all three aponeuroses pass in front of the muscle. The lateral margin, or linea semilunaris, extending from the ninth costal cartilage above to the pubic tubercle below, marks the level of this blending of aponeuroses above the umbilicus but the external oblique passes somewhat medial to this below the umbilicus before fusing with the underlying aponeurosis. A pararectal incision below the umbilicus, therefore, divides two layers before the rectus muscle is displayed.

Contained in the Rectus Sheath are the superior and inferior (deep) epigastric vessels, which occasionally rupture as a result of trauma, giving rise to a hæmatoma, which may spread from costal margin to symphysis. The Rectus muscle is inserted into

the fifth, sixth, and seventh costal cartilages and the ensiform cartilage and it presents three, or occasionally four, tendinous intersections attaching it closely to its anterior sheath. These are at the level of the ensiform cartilage and umbilicus, midway between the two and midway between umbilicus and symphysis, where a fourth one may be present The two muscles are separated by the *linea alba*, a tendinous band stretching between ensiform cartilage and symphysis pubis. It is formed by the intersection of the aponeurosis of the oblique and transversalis muscles and is narrow below, where the interval between the muscles is minimal. Above, where the muscles diverge, it is broader and presents interstices through which fat may herniate (epigastric hernia). The linea alba is rendered tense by the Pyramidalis muscles, inserted into it midway between umbilicus and symphysis pubis, passing in front of the Rectus muscles from their origin on the front of the pubic bone. The muscles are, therefore, contained in the Rectus Sheath, which also contains the terminal portions of the lower six intercostal nerves.

The *External Oblique* muscle arises from the lower eight ribs, interdigitating with the slips of origin of the Serratus Anterior and Latissimus Dorsi—a point of some importance in the performance of radical mastectomy. It is the largest of the three lateral muscles, having a free posterior, muscular border running vertically downwards from the last two ribs to the anterior half of the outer lip of the iliac crest. The remaining fibres become aponeurotic at the linea semilunaris and are directed downwards and inwards, the lower margin extending from the anterior superior spine to the symphysis and forming the inguinal ligament. The anatomy of this region is therefore discussed in relation to hernia.

Above the iliac crest the External Oblique is crossed obliquely by the fibres of the Latissimus Dorsi, and the triangle so formed is known as the lumbar triangle of Petit. A lumbar hernia, or occasionally a tuberculous abscess from the spine, may present at this point

The *Internal Oblique* muscle lies under cover of the External Oblique and arises from the outer half of the inguinal ligament, the anterior two-thirds of the middle lip of the iliac crest and the lumbo-dorsal fascia. It is inserted by its posterior fibres into the inferior border of the lower three ribs, being continuous with the Internal Intercostal muscles. The fibres arising from the inguinal ligament, paler in colour than the others, form the conjoined tendon with the corresponding part of the Transversalis.

The *Transversalis* (or Transversus) muscle lies deep to the Internal Oblique and arises from the outer third of the inguinal ligament, the anterior three-quarters of the inner lip of the iliac crest, the inner surfaces of the lower six costal cartilages, interdigitating with the Diaphragm, and from the lumbo-dorsal fascia. It is inserted into the conjoined tendon and the linea alba, forming the Rectus Sheath, and is aponeurotic throughout the insertion. The upper muscular fibres, however, reach within an inch of the linea alba in the posterior Rectus Sheath.

The *lower six intercostal nerves* supply the musculature of the abdominal wall, entering between the digitations of the diaphragm and Transversalis muscle and lying between the latter and the Internal Oblique as far as the outer border of the Rectus Sheath, which they pierce. Entering the muscle on its posterior aspect, at about the junction of its inner third with the outer two-thirds, they finally emerge as anterior cutaneous branches close to the linea alba. The skin over the ensiform cartilage is supplied by the sixth, the umbilicus by the tenth, and the symphysis pubis by the twelfth.

The seventh and eighth nerves incline obliquely upwards and supply the upper third of the abdominal wall, entering the Rectus close to the ninth costal cartilage. The ninth and tenth nerves are nearly horizontal and supply the middle third and the eleventh, subcostal, ilio-hypogastric and ilio-inguinal supply the lower third. The last two are branches of the first lumbar nerve, supplying muscular twigs to the Internal Oblique and Transversalis and cutaneous branches to the inguinal and genital region.

ABDOMINAL INCISIONS

The siting of abdominal incisions is of the utmost importance and certain well-tried examples have stood the test of time. They have in common:

(1) Good access to the part required.
(2) Minimal damage to nerves in the abdominal wall.

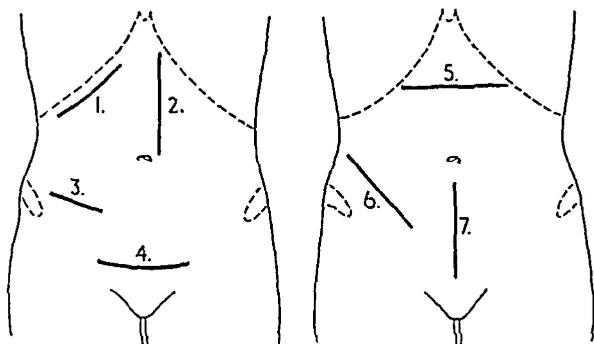


Fig. 1. Abdominal incisions.

1 Subcostal (Kocher's)
2 Paramedian (left)
3 Grid-iron (crease)
4 Pfannenstiel
5 Transverse
6 Rutherford Morison (muscle cutting)
7 Lower midline

(3) Division of muscles in the line of their fibres.
(4) Division of muscle in preference to aponeurosis, where one or other is necessary.
(5) Skin incisions in creases, where possible.
(6) Avoidance of drainage through the main wound, tubes being brought out through separate stab incisions.
(7) Adequate length, to minimize the use of retractors at operation.

Individual preference naturally plays an important part in the choice of a surgeon's incision and the development of relaxants in anæsthesia has done much to equalize the merits of respective methods of approach. It is also pertinent that speed is a much less portant factor now than in the days of chloroform and ether.

MEDIAN INCISIONS

The mid-line incision above or below the umbilicus has waned in popularity, largely because it gives the highest incidence of dehiscence and post-operative hernia. This is because the scar in the linea alba stretches, particularly in the upper abdomen, and a bulge is then inevitable, especially where the Recti are widely divaricated. Careful suture of such an incision, even with wire, does not exclude this possibility and the close apposition of the linea alba to the transversalis fascia and peritoneum may really mean that only one good layer of sutures can be placed. That these should be interrupted and not continuous goes without saying, and the same applies in general terms to all suturing of muscle and fascia in the abdominal wall.

The median incision below the umbilicus can easily transgress one of the Rectus Sheaths, as the linea alba is so narrow. The underlying transversalis fascia and peritoneum, however, are easily separated and can more readily be sewn as a distinct layer than in the upper abdomen

Drainage through this type of incision, which has been largely employed in the acute abdomen, only increases the chance of herniation.

THE PARAMEDIAN INCISION

This invôlves division of the rectus sheath 1 in. or so to either side of the mid-line, retraction of the muscle laterally and division of the posterior sheath (above the semilunar fold) *in the same line as the anterior. In order to retract the muscle, however, one* or more tendinous intersections must be freed from the anterior sheath and care must be taken to tie off the little vessel usually found with them. More important than these are the deep epigastric vessels lying behind the muscle and within its sheath. These may be traumatized, if the muscle is too forcibly retracted laterally, and a hæmatoma running the whole length of the sheath result

Division of the Rectus muscle in its inner third, splitting its fibres in the line of the incision, is practised widely as a modification of this incision. It was advocated by Mayo Robson for cholecystectomy and an inward ("hockey stick") angulation of the upper end towards the ensiform cartilage may be added to it.

It is important in splitting the muscle in this way not to do it too laterally, to avoid damage to its main nerve supply. Even the most medial division divides the terminations of the intercostal nerves and may lead to atrophy of some fibres of the muscle.

The paramedian incision, however, is a strong one and it can be extended at will. On this account, it is particularly useful for exploration, where the diagnosis is in some doubt. In peritonitis of unknown origin, for example, a small incision half above and half below the umbilicus may be extended radically upwards or downwards, when the cause has been found by the exploring hand.

THE PARARECTAL INCISION

This is also known as Battle's incision and is most often used in appendicitis. Though suitable in dealing with a pelvic appendix, it is not popularly employed. The Rectus is displaced medially after its sheath has been incised in a line parallel and just medial to the linea semilunaris. The intercostal nerves should be carefully identified before incising the posterior sheath and care must be taken of the epigastric vessels. The objection to the incision is the danger of injuring nerves, if it has to be extended

As in the mid-line wound, drainage through it is undesirable. The tube tends to prevent the muscle from covering the scars in the anterior and posterior sheath. The valvular action is thus impeded and one of the main advantages of the incision lost.

THE GRID-IRON INCISION

McBurney's incision for appendicectomy is the classical example. It crosses a line from the anterior superior spine to the umbilicus at the junction of its outer and middle third. This is about 1½ in. medial to the anterior superior spine in an adult. One-third of the incision lies above this line and two-thirds below. The External Oblique is then incised in the same line and the Internal Oblique and Transversalis at right angles—all in the line of their fibres. The Transversalis fascia and peritoneum are divided in the same line as the skin.

The procedure can be modified by making the skin incision in the crease (Lange's line), as the resulting scar is better. Its main disadvantage is that it gives limited access and can only be extended by carrying it into the Rectus sheath. This immediately negatives its main advantage of being valvular and endangers the ilio-hypogastric nerve. The resulting weakness of the conjoined tendon, which it supplies, predisposes to the development of direct inguinal hernia.

The grid-iron incision may also be employed on the left side for the performance of an inguinal colostomy. The steps are identical and the direction of the skin incision of less moment, as an ellipse of skin should be excised.

MUSCLE-CUTTING INCISIONS

Oblique incisions through the flat muscles have been popularized by Rutherford Morison and others. They give particularly good access to the cæcum and ascending colon on the right and the pelvic colon on the left. All the muscles are divided in the line of the skin incision, the Internal Oblique and Transversalis thus being divided at right angles to the direction of their fibres. Extension into the Rectus sheath will allow of retraction or even transverse division of the muscle, a procedure which does not weaken it, as it passes between adjacent nerves without injuring them. The peritoneum may be stripped medially without opening it and access to such structures as the upper two-thirds of the ureter or the sympathetic chain very satisfactorily obtained.

TRANSVERSE INCISIONS

These have increased in popularity in the upper abdomen, particularly for such procedures as pancreatectomy and total gastrectomy. The Rectus muscles are divided transversely and sutures may be passed into the muscle through the anterior sheath before dividing it, in order to prevent retraction of the fibres. An elliptical incision bowed upwards to the ensiform cartilage at its mid-point is a useful modification of the horizontal incision in operations on the pancreas. These incisions heal well and give good scars but do not, of course, allow of good access to the lower abdomen if any additional manœuvre is required there.

Rutherford Morison employed a right sided transverse incision from a point 1 in. below the tip of the twelfth rib to the middle line, a third of the way from the ensiform cartilage to the umbilicus in recurrent operations on the biliary apparatus, where previous procedures have been carried out through a Kocher or paramedian incision Definition

of the liver edge can be carried out as an early step and the origin of the diaphragm can be incised to give good access, perhaps by placing the incision a little higher. Thoraco-abdominal incisions, starting in an intercostal space and carried forwards through the costal margin to become transverse, upper abdominal incisions are particularly useful in œsophago-gastrectomy on the left side and rarely for exploration of the porta hepatis, or porto-caval anastomosis, on the right.

The *Kocher* (subcostal) oblique incision is a modified transverse incision, used particularly for operations on the gall bladder in obese subjects and those with a wide costal angle. The incision, dividing the Rectus 1 in. below the costal margin between the eighth and ninth intercostal nerves if correctly placed, is liable to damage the ninth and tenth nerves if extended laterally. On the left side, a similar incision may be employed for splenectomy.

A transverse incision below the umbilicus in a skin crease (Pfannenstiel) is employed in gynæcology, as the resulting scar is a good one. The linea alba is incised vertically and the Recti retracted. This, of course, gives good access to the retropubic space and has also been used for operations on the bladder and prostate, especially in the Millin prostatectomy. Post-operative hernia is far less common than after vertical suprapubic incisions.

ABDOMINAL INJURIES

The vast field of abdominal injury claims high priority in war. But the growing complexity and speed of modern methods of transport brings in its train a steadily increasing number of accidents involving abdominal trauma in peace time. In some countries, too, the classical sword thrust and bullet wound have diminished little, if at all, in frequency and the emergency surgeon quickly acquires experience of them.

It is important, therefore, to formulate some plan regulating their management and to realize that, penetrating or otherwise, abdominal injuries are full of traps for the unwary and disappointment, sometimes where it is least expected. In doubtful cases, indeed, the risk of laparotomy may well be less than that of observation but this is no reason to subject a patient to it, who can recover more rapidly without This applies only to closed injuries of solid viscera or those of the abdominal wall; all others require exploration and toilet of wounds of entry and exit, as in other parts of the body.

DIAGNOSIS

General principles governing the diagnosis of abdominal injury are closely connected with the problem of surgical shock and the effects, in particular, of internal hæmorrhage. *This especially concerns the officer in charge of triage and resuscitation in dealing with* large numbers of casualties, for men with abdominal injuries rank high in priority and profit by surgery at the earliest moment at which they are fit for it. The time interval since receipt of injury is, in fact, the most important single factor in prognosis but time spent on pre-operative resuscitation may be imperative to a successful outcome.

The history is usually brief and obvious but the nature of the missile and position of the patient at the time of wounding are both of great importance Neither may be known and they are clearly of less significance in closed than in open wounds. Coincident injury, also, has a bearing on the problem, for abdominal injury with a severe head

injury, or limb injury requiring amputation, is more often than not fatal. Exposure since injury is also an adverse factor and any form of journey to a surgical centre injurious to such a patient.

The first duty of the surgeon receiving a patient with an abdominal injury is rapidly to assess, with as little disturbance to the patient as possible, whether an operation is required and, if so, how soon it can be done. Rigidity of the abdomen, signs of internal hæmorrhage, lack of intestinal sounds on auscultation and radiological evidence of gas under the diaphragm may all be sufficient to indicate the need for exploration. But only the last of these is an absolute criterion. Rigidity, signs of shock not due to internal bleeding and absence of intestinal sounds can all occur in severe contusions of the abdominal wall, fractures of and injuries to the spine and thoracic or thoraco-abdominal wounds. Care must be taken, therefore, where the injury is not a penetrating one, to detect deterioration due to hæmorrhage or progressive shock in spite of measures of resuscitation and to realize that these demand surgical interference. In penetrating injuries of all types the need for operation is obvious from the start and only the timing requires judgment. In general terms, a systolic blood pressure over 90 is desirable before surgery is undertaken, but delaying laparotomy in rare instances, where this cannot be achieved, may amount to a death sentence. Profuse bleeding from the mesentery in multiple injuries of the small intestine provides examples of this type and they are relatively easy to recognize.

Pallor, the facies of shock and vomiting in the ambulance on the way to hospital are usual features of the patient with a hollow viscus injury and X-ray will not be required, where intestinal injury is obvious. In doubtful cases, however, it is invaluable as, for example, in wounds of the buttock, where penetration of the peritoneum and bowel is in question or injuries of solid viscera, where coincident hollow viscus injury has to be excluded. Eventration, likewise, will render X-ray unnecessary and it is quite surprising how little intestinal damage may be found in this type of injury. The wound through which the intestine has protruded is often quite small and peritoneal soiling conspicuously slight. With large wounds, of course, where bowel has prolapsed through a gross deficiency, the converse is the case.

PRE-OPERATIVE PREPARATION

The paramount importance of blood in preparing these patients for operation cannot be over emphasized. This single factor alone was largely responsible for the improvement in the figures of abdominal surgeons in the 1939–45 War over those of their predecessors in 1914–18. Analysis of the cause of death in the 20–25 per cent fatal cases shows blood loss as far the most potent factor, with peritonitis and ileus as comparative rarities. Death within 48 hours of injury, in fact, accounts for the great majority of fatalities and bleeding from the mesentery is the commonest single cause.

Plasma is a poor substitute for blood in dealing with these patients and the danger of homologous serum jaundice a real one. Other substitutes such as dextran and plasmosan have no more to recommend them. It is important, therefore, where intraperitoneal bleeding is suspected, *to have the means of transfusion at hand*, if not in progress, and two simultaneous administrations may be necessary, where blood loss has been severe and rapid replacement is required. A Ryle's tube will be of material assistance to the anæsthetist and it should be an inviolable rule that a specimen of

urine has been obtained, if necessary by catheter. Only in this way can damage to the urinary tract be excluded, occurring as it does in a large variety of wounds, even where the paths of entry and exit seem to render it unlikely.

Finally, it behoves the surgeon to take into account possible rib damage in injuries of the liver and spleen, fractures of the transverse processes and bodies of the lumbar vertebræ in kidney injury and involvement of the bony pelvis in buttock wounds and those of the bladder and rectum. Appropriate X-rays may, therefore, be of considerable assistance at operation but their value must always be offset against the disturbance to the patient required in obtaining them and the quality of radiography with the apparatus available. Portable films, for example, may be the only form obtainable and these are of limited value.

Anæsthesia. Patients with abdominal injuries are commonly young or middle-aged men in previously excellent health, who stand surgical shock and anæsthesia well. In children and the older age groups, however, the same is not true and powers of recovery are less. Modern methods of anæsthesia have much to offer here and the employment of positive pressure in thoraco-abdominal wounds to maintain lung inflation, use of relaxants of the curare group to diminish the dose of irritant agents, value of controlled respiration in combined thoracic and abdominal procedures and universal application of laryngeal intubation are some of the outstanding contributions. Bronchoscopic suction is also generally accepted as a useful manœuvre at the end of a long procedure, particularly where bronchial secretion during anæsthesia has been considerable.

Local anæsthesia has its advantages, where intraperitoneal injury is not suspected and at the extremes of life, but, in the author's view, is time-consuming and contraindicated in the management of gunshot wounds. Spinal anæsthesia likewise has nothing to recommend it, as hypotension from blood loss is already a feature in serious cases and this is likely to augment it.

SOLID VISCUS INJURY

The Spleen

Rupture of the spleen occurs most commonly as the result of a fall from a height or severe blow over the lower costal margin on the left side. Damage to the ninth, tenth, or eleventh ribs is often coincident and renal injury may occur at the same time. The twelfth rib, if abnormally long, may also be fractured. A kick in the loin from the boot of an enthusiastic wing forward has been responsible and mishaps, such as falling off a horse or bicycle against some solid object, and compression injuries between moving vehicles and a wall have all produced rupture of the spleen.

If it is already enlarged (e.g. in malaria) much less violence is required and examples are recorded in which the trauma was so slight as to pass unrecognized at the time. Delayed rupture, where several days may elapse between injury and intraperitoneal bleeding, is explained by late rupture of the capsule, which contains a hæmatoma for a number of days and finally gives way from pressure necrosis. Indirect injury, such as a sudden twisting movement, may be sufficient to produce this result

Apart from the two types of injury mentioned, the spleen is commonly found injured in thoraco-abdominal wounds on the left side. It may even prolapse externally or through the rent in the diaphragm It is very easily removed through the chest, as control of the splenic artery in the lieno-renal ligament is easy from above.

The signs and symptoms of rupture of the spleen are usually not difficult to recognize, rigidity being especially marked over the left upper Rectus muscle. Pain in the left hypochondrium, bruising of the abdominal wall and loin, shock and signs of rib injury are cardinal. Rarely a pneumothorax is also present when a bone fragment in a penetrating wound projects through the pleura. Pain in the left shoulder aggravated by breathing, is also relatively common. A rising pulse rate and the signs of free fluid within the abdomen will then rapidly indicate the need for exploration and splenectomy.

A left paramedian incision will allow of adequate exposure and permit inspection of the other abdominal contents. A subcostal incision may be preferred, where the spleen was known to be normal before injury and no abdominal incision at all will be required when splenectomy is part of a thoraco-abdominal exploration. The combination of a vertical mid-line incision with a transverse cut across the left Rectus muscle combines the disadvantages of both and is particularly liable to lead to subsequent hernia. Only where the spleen is large and fixed by perisplenitis and old infarction to the diaphragm does this method of approach seem justifiable.

Injury to the tail of the pancreas must be carefully guarded against during operation and it is wise to remove blood and blood clot from the abdomen by suction as far as possible. The very rare instances in which the spleen has only sustained a shallow guttered wound in the course of a missile, entering the left pleural cavity and penetrating the diaphragm, have provided an occasional example of suture without removal but splenectomy is, for practical purposes, the treatment of a ruptured spleen. Post-operative pyrexia is a usual feature of the course of these patients and wound dehiscence is particularly liable to occur. The sutures should not, therefore, be removed for approximately 9–10 days. The question of drainage at operation depends primarily on the presence or absence of associated visceral injury and the efficacy of removal of blood from the peritoneal cavity.

The Liver

Much has been written on the subject of injuries of the liver, which usually result from direct injury. It is almost invariably involved in right sided thoraco-abdominal wounds and does much to protect the remaining abdominal viscera. For this reason alone, right sided thoraco-abdominal wounds carry a better prognosis than left sided ones and some, like the closed injuries, can be treated conservatively. When very gross trauma to the liver has been sustained and hollow viscus injury is coincident, the prognosis is grave

Wounds of the liver notoriously carry with them the risk of biliary peritonitis but hæmorrhage is a greater danger It is here that transfusion is so vital and this alone may make operation possible Through-and-through sutures placed with round bodied needles and tied over omentum, omental grafts, gelatin sponge, oxycel and other similar preparations may be of considerable help, but the use of gauze packs is only to be deprecated. It is also important to remember that bleeding from the liver shows a tendency to spontaneous arrest and to appreciate the fact that friability and difficulty of access may make suture of certain wounds more hazardous than non-operative measures. Drainage is naturally essential, when laparotomy or thoracotomy has been undertaken and the resulting biliary fistula may take long to heal. The retention of metallic foreign bodies in the substance of the liver has, however, very little bearing on this problem and

they may, when small, remain for years without any effect. Larger fragments, sometimes by reason of the presence of contaminated clothing or debris carried in at the same time, may cause abscesses, with pyæmia as a possible complication.

The non-operative management of smaller wounds of the liver, already showing a tendency to stop bleeding, necessitates close and careful watch on the pulse rate, morphia, antibiotics to control the peritoneal reaction from bile and blood, and transfusion where indicated by a progressively falling hæmoglobin reading.

The Kidney

Rupture of the kidney is one of the most frequently encountered injuries to abdominal viscera. It rarely requires nephrectomy and should almost invariably be treated conservatively for 48 hours before exploration. The tear is in the transverse axis and nephrectomy is only likely to be necessary where damage to the pedicle has been sustained. Indirect injury from sudden bending and twisting movements is rarely responsible and blows and falls on the loin are the predominant cause. Gunshot and stab wounds of the back and thoraco-abdominal wounds include a number with renal involvement. The liver and spleen may also be implicated.

The cardinal sign of this injury is hæmaturia, which may only be evident in a shocked patient after the passage of a catheter. Clot retention has occasionally resulted and a Bigelow's evacuator may be required to clear the bladder before lateralization of the renal bleeding by cystoscopy is possible. This, of course, is the first requirement in investigating a case of rupture of the kidney. The second is to determine the presence of a second kidney and gain some idea of its functional efficiency. Five cubic centimeters of indigo-carmine (0·4 per cent) intravenously should appear at the ureteric orifice in 5–8 minutes, but this is not as reliable as a pyelogram. Pyelography, however, plays an unimportant part in the management of renal injuries, as diagnosis and treatment are usually quite simple without it.

The fact that the blood is going down to the bladder in this condition implies that the pelvi-ureteral junction is not occluded by clot. Very sudden cessation of a severe hæmaturia may indicate this state of affairs and be followed by deterioration in the general condition, demanding exploration without further delay.

Coincident hollow viscus injury, of the duodenum or colon in particular, makes the outlook more serious but the development of antibiotic therapy has made it possible to preserve even the kidney contaminated with colon contents. Before the advent of streptomycin and its successors, combined renal and colic injury on the left side nearly always demanded nephrectomy

Exploration of kidney wounds by the transperitoneal route is required, where intraperitoneal injury is suspected in addition. This of course allows of manual palpation of the other kidney and rapid control of the pedicle in cases of severe bleeding. Recovery has even been described after avulsion of the kidney from its pedicle, the organ being found free in the peritoneal cavity.

Damage to the transverse processes of the lumbar vertebræ is not uncommon as a result of hyperflexion by the Psoas muscle, which goes with this injury, and it must be borne in mind in relation to after treatment. Other bone (e.g. rib) injury may also be relevant. The most important consideration, however, is the presence of red cells in a centrifuged deposit of the urine. The patient should not be allowed up as long as these

are present and three weeks or even more may occasionally elapse before they completely disappear. Ambulation before this carries the risk of secondary hæmorrhage and even delayed rupture of the kidney.

The danger of extrarenal spilling of pyelosil has been adduced as a contraindication to intravenous pyelography but this is very slight. After the initial shock has passed off, it is a useful means of assessing what renal damage, if any, is likely to be permanent. In general terms, however, healing is remarkably complete and infarcted or non-functioning areas in the kidney exceptional.

The Pancreas

This is the only other retroperitoneal viscus demanding consideration in dealing with abdominal trauma. Crushing of the upper abdomen, as between the tailboard of a reversing lorry and a wall, or between a carriage and buffers, may rupture the pancreas and be rapidly fatal from coincident damage to the suprarenals, cœliac axis, aorta or inferior vena cava. In less serious injuries recovery may occur and a traumatic pseudocyst of the pancreas result. This will ultimately require exploration, when simple drainage may result in pancreatic fistula and some form of anastomosis to the jejunum be preferable.

Bleeding from the pancreas in penetrating wounds is liable to be severe but it can often be controlled, if the operation is undertaken soon after receipt of injury. Coincident hollow viscus injury and the development of the so-called "retroperitoneal syndrome" associated with some of them are then the chief factors militating against recovery. The retroperitoneal cellular tissue infection or hæmatoma, contaminated, for example, by colon contents, is obviously a dangerous complication from its extent and the difficulty of adequate drainage. The term "syndrome" for this state of affairs seems, however, unnecessary and its terrors have been somewhat lessened by the advent of antibiotics.

HOLLOW VISCUS INJURY

This group comprises the majority of abdominal wounds, especially of the penetrating variety. The solid viscera to some extent gain protection from the skeleton, under cover of which they lie, the liver, spleen, and kidneys all being related to ribs and the pancreas to the vertebral column. The great strength and thickness of the muscles of the back and buttock render wounds inflicted on this region less serious to abdominal viscera than those of the thinner abdominal wall. Though the bony pelvis and lower thoracic cage afford some measure of protection to bladder and rectum below and stomach, in particular, above, the whole length of the small and large intestine is exposed behind the abdominal wall

In general terms it is stated that wounds over the Rectus muscles involve the small intestine and over the Obliques both small and large. This, however, is only a generalization, as the former may frequently affect the transverse colon and the latter no loop of small bowel or even colon but the kidney and retroperitoneal region

The signs and symptoms of these injuries are usually easy to recognize and emphasis has already been laid on the paramount importance of blood loss from the mesentery. Peritoneal contamination is less important but clearly depends, above all, on the site of injury, being greatest in the pelvic colon, where the bowel contents are most heavily infected. Mesenteric bleeding, conversely, is most serious in the small bowel, where the

contents are not infected. The distinction between the wounds of the small and large intestine determines the great difference in their treatment, resection or suture without exteriorization being the rule in the small intestine and suture with or without exteriorization the guiding principle in the large. We may go even further and state that exteriorization should be avoided in dealing with the right side of the colon and employed in cases of doubt on the left side. The antibiotics, and streptomycin in particular, have brought about this new attitude to large bowel injury and the importance of sulphasuxidine is hard to exaggerate. Both should be used as a routine in colon injury and temporary proximal colostomy or exteriorization employed only when satisfactory suture cannot be achieved. In buttock wounds involving the rectum, where suture of the bowel is difficult and adequate drainage impossible, a left inguinal colostomy as a temporary measure may be a life-saving procedure.

Operative Treatment. Some of the important principles governing the management of intestinal injuries have already been elucidated but they are secondary in importance to decisions relative to the time of operation and pre-operative resuscitation. Morbidity and mortality begin to rise when more than 4–6 hours elapse between wounding and definitive surgery, and blood transfusion is by far the most important single factor in recovery from shock. Once this has been achieved, laparotomy is imperative in hollow viscus injury and certain general rules may be stated as important in carrying it out.

(1) Though entry and exit wounds should be excised, they should, if possible, be excluded from the main wound of exploration.

(2) A paramedian wound is the most useful when the wound of entry lies within the linea semilunaris.

(3) Wounds of the iliac fossæ may be explored through a muscle-cutting, oblique incision but the paramedian incision still has great advantages.

(4) Location of metal fragments is not an important part of abdominal exploration for trauma.

(5) The whole problem must be defined before any part of it is undertaken. This may result in resection of a loop of small bowel as a quicker and more effective operation than suture of multiple perforations.

(6) A rigid plan of inspection must be adopted, searching the intestinal tract from duodeno-jejunal flexure to recto-sigmoid junction. Uninjured bowel must be returned to the peritoneal cavity and not left outside, while suture of perforations is being undertaken

(7) Single layered suture is permissible in the small intestine It is rare, however, for the saving of time to be so important that a second layer cannot be completed.

(8) Exteriorize the left side of the colon when in doubt

(9) Adequate suction of blood and contaminated contents from the pelvis is the best form of prophylaxis against subsequent residual abscess Drainage is often wise.

(10) Laparotomy, when in doubt as to diagnosis, may often be safer than expectant treatment.

Hollow viscus injury in the absence of penetrating or perforating wounds tends to be confined to the fixed portions of the small intestine, i.e. duodeno-jejunal flexure and terminal ileum T[illegible]ansection is, indeed, not uncommon as a consequence of closed injury, when a severe blow is borne against the tensed abdominal wall. Severe shock, signs of

internal hæmorrhage, vomiting and a rigid and silent abdomen will soon be found on examination of the patient and delay in laparotomy should be as short as possible. Where small intestine injury, such as that described, is not found, perforation of the less mobile portions of the colon may have occurred. The principles to be observed in treating it are, of course, similar to those governing the management of open wounds

RESULTS

The main factors affecting prognosis in abdominal injury are the age of the patient, nature and severity of the wounding agent, interval between receipt of injury and operation and availability of blood and restorative measures in the treatment of shock before operation is undertaken. Associated injuries, particularly of the thorax and its contents affect the outlook considerably and injuries of the pelvic girdle and buttock with damage to the rectum, urethra or bladder are notoriously dangerous. In spite of all these considerations, an overall mortality of less than 20 per cent should be attainable. This may be judged from figures of published series of war wounds and experience in large hospitals, in areas where industrial accidents are common and abdominal trauma not infrequent.

INTESTINAL FISTULÆ

Fistulous communications between the abdominal viscera and the abdominal wall occur in all parts of the alimentary canal. They follow trauma or surgical operations but are more commonly the result of an inflammatory process or growth, starting in a hollow viscus and invading the overlying parietes. A fistula has, thus, to be distinguished from a sinus, which is a track leading down from the abdominal wall but not demonstrably connected with an abdominal viscus. Gas and intestinal contents are, of course, discharged from a fistula—called fæcal, in the large or small intestine; pus, on the other hand, comes from a sinus, as, for example, in actinomycosis. This disease, affecting the ileocæcal angle, is often complicated by multiple sinuses. Intestinal fistulæ, by comparison, are more often single.

The effects of an intestinal fistula depend primarily on its site, the results of dehydration and fluid loss, which it entails, and the changes induced on the surrounding abdominal wall by digestive juices and enzymes in the escaping fluid. These may so complicate the picture as to be fatal before the primary disease process can be arrested or cured. The principles governing their management must, therefore, be clearly understood in order to minimize these effects as far as possible. Before discussing them, examples in individual viscera may be considered.

(1) THE STOMACH

Gastric fistula is historically famous from the story of Alexis St. Martin in 1822, who sustained at the age of eighteen, by the accidental discharge of a musket, a thoraco-abdominal wound on the left side, with herniation of the lung and stomach. He survived more than 60 years and, for many months, wore a tent-compress and bandage to control the loss of food and drink. Extrusion of the mucosa, however, in valve-like fashion, rendered this unnecessary after a time and an opening about an inch in diameter remained, which could easily be depressed by the finger. The man took ordinary food and drink

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of feeding a patient [illegible]. It has been used in the treatment of carcinoma of the [illegible] growths has been a practicable proposition. [illegible] gastrotomy is avoided as far as possible.



Fig. 3. External gastro-colic fistula

(2) THE DUODENUM

Duodenal fistula is a grave and often fatal sequel of gastrectomy, where difficulty has been experienced in infolding the duodenal stump. This may be the direct result of ulceration, where penetration of the pancreas has been extensive, and particularly where the process has spread beyond the first part of the duodenum. Experience shows that gastrectomy in some of these cases without ablation of the ulcer is the most reliable way of avoiding this disaster.

Other examples of a fistulous track leading to the duodenum are found rarely after transduodenal exploration of the ampulla of Vater, but biliary and pancreatic fistulæ are both beyond the scope of this discussion.

and worked for 11 years, as a servant, in the house of Dr. Beaumont, who described the case history in detail.

Ulceration and carcinoma of the stomach are much commoner causes of gastric fistula to-day. Adhesion to the abdominal wall and perforation may occur spontane-

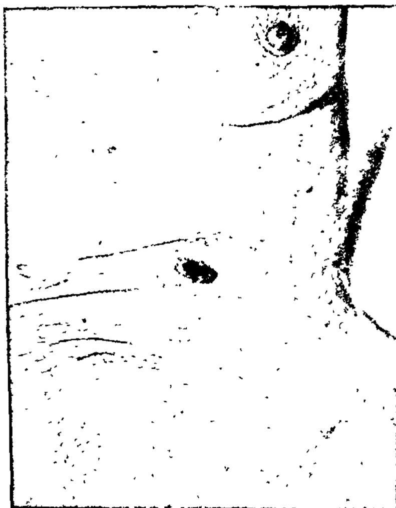


FIG 2. Gastric fistula (gastrostomy)

ously, but are more usually the sequel of an operation. Such was the case in the fistula shown (Fig. 2), which occurred in a lady of 65, who had had previous operations for perforated gastric ulcer, hour-glass stomach and, finally, linitis plastica with gastro-colic fistula. Radical resection proved impossible and an external fistula resulted.

Internal gastro-colic fistula or gastro-jejuno-colic fistula is commoner and results from invasion of the colon by a growth in the stomach or perforation of a stomal ulcer into the colon after gastro-enterostomy. Infiltration of the stomach by a growth in the colon is a very much rarer occurrence

Gastric fistula, finally, may be the result of attempted suture, of a very large gastric ulcer, breaking down and discharging through the abdominal wound. Alternatively, a fistula may be deliberately made surgically by gastrostomy (Fig. 2) as a temporary means

Fistula on the left side of the colon is commonly due to diverticulitis and its complications. Pericolic abscess may track into the abdominal wall or thigh and internal fistula (e.g. vesicocolic) is also common. Drainage of pelvic abscesses associated with colon disease may also produce a fæcal fistula in this way. It is important, therefore, that this complication should be borne in mind when abscesses of the abdominal wall are encountered on the left side. The presence of gas on an X-ray may be indicative in this connection. Finally, associated fistula-in-ano occurs with some inflammatory diseases of the colon and rectum, and even with regional ileitis, but the subject is dealt with under a separate heading.

Principles of Treatment

The first essential in dealing with a patient with an intestinal fistula is a correct appraisal of its cause. The history is naturally of paramount importance and it is often true, as with fistulæ in the urinary and biliary tract, that obstruction beyond the level of the fistula is the primary cause of its appearance.

Radiological studies may, therefore, be very helpful, in the form of sinograms, with an opaque, innocuous medium such as lipiodol to outline the fistulous track, or barium by meal or enema. Simpler tests, such as the administration of charcoal or carmine by mouth, may also be useful at times in establishing the presence of a fistula with certainty. Rarely, additional evidence may also be obtained by simple analysis of the reaction or enzyme content of the discharge from a wound. A strongly alkaline reaction, for example, will be detected in a duodenal fistula, whereas a gastric one will be be acid. The former, likewise, will have pancreatic ferments and bile in the discharge, which will both be absent when the fistula is proximal to the pylorus.

The important aspects of the management of intestinal fistulæ, once their nature has been established, are as follows:

(1) Suction. As a general rule, an intestinal fistula is a source of considerable disturbance to a patient and frequent dressings are often required. The need for these can often be considerably reduced by continuous suction with a silent motor, if possible, or negative pressure in some form, if a motor is not available.

A catheter or tube in the fistulous track may facilitate this process and one of the uses of a sinogram is in allowing such a tube to be placed in the most advantageous position.

Another important consideration in maintaining suction drainage is that it should be instituted at the earliest possible moment and kept up continuously until it is no longer required.

(2) Protection of the Surrounding Skin. This is a vital requirement, in which suction drainage, where possible, is of inestimable value. Rarely, however, is a tube skin-tight and some leakage therefore occurs. The higher the fistula in the alimentary canal the more noxious is the discharge on the skin. In a fæcal fistula on the left side of the colon, for example, skin excoriation is unusual; in a duodenal fistula it is extreme.

Basic protection of the skin by oily and greasy preparations in ointment and liquid form has certain advantages but these may interfere with the fitting of belts and appliances. Aluminium paste (Baltimore paste) is one of the best known but it is difficult to apply satisfactorily unless well made and at the right temperature. Vaseline, paraffin bases, zinc ointments, iron filings, soluble rubber or latex solution, Innoxa barrier

The very rare duodenal fistula following right nephrectomy and due to accidental injury to the third part of the duodenum carries a better prognosis than the first two groups.

(3) THE SMALL INTESTINE

The establishment of a fistula into the jejunum by *jejunostomy* is an accredited and important surgical exercise, employed as a method of nourishing a patient with an œsophageal or gastric lesion, precluding satisfactory oral feeding. It may even enable a subsequent radical procedure to be carried out. Carcinoma of the œsophagus and large peptic ulcers, occasionally with a gastric fistula, may be handled in this way but the patients, in whom this course of action is indicated, are few and so dehydrated and ill that this form of palliation often only delays the final issue. In œsophageal carcinoma, moreover, it should be borne in mind that a jejunostomy adds a little to the technical difficulty of extirpation, if done high and if followed by œsophago-jejunal anastomosis.

Ileostomy contrasts very sharply with this, being employed almost exclusively in the treatment of ulcerative colitis. Terminal ileostomy is much preferable to ileostomy in continuity but this is unimportant in relation to small intestine fistulæ, except in so far as it affects appliances used to control them. An ileostomy in continuity in the treatment of paralytic ileus has virtually been abandoned since the introduction of Miller-Abbott and other forms of suction drainage.

A fistula into the ileum, apart from those already mentioned, occasionally follows operations for appendicitis, where damage to a loop of intestine is sustained, and operations for resection of the small intestine, as in obstruction by regional ileitis or strangulated hernia, where a coil of bowel becomes adherent to the laparotomy wound and discharges through it a few days afterwards. Secondary operation by anastomosis to exclude the fistula, or further resection, is often required here.

FÆCAL FISTULA

This is by far the most important group of cases to be considered amongst intestinal fistulæ and comprises quite a number, apart from patients in whom cæcostomy, transverse or inguinal colostomy is done as a step in treating malignant or inflammatory obstruction of the large bowel. This is considered elsewhere but it may be stated that a growing preference for decompression in the transverse colon over cæcostomy carries with it more efficient defunctioning of the distal bowel and a fistula much easier to manage In general terms, fæcal fistula gets easier to control, the nearer it approaches the pelvic colon.

Fæcal fistula following operations for appendicitis and appendix abscess deserves special consideration and the causes may be briefly considered. When it occurs, it is often seen within 48 hours of operation and its appearance may coincide with a marked improvement in the patient's condition This, of course, depends on the cause, necrosis of the cæcal wall in the region of the base of the appendix being an important one. Other possible factors in the development of such a fistula are pressure necrosis from a drainage tube, failure to find a fæcolith, failure, probably deliberate, to remove the appendix, damage to the cæcum or a coil of bowel in the wall of an abscess, associated disease (e.g carcinoma) in the cæcum or ileum (e g regional ileitis) and, finally, the presence of a foreign body.

skin-grafted In intestinal fistulæ, however, exteriorization of the bowel has rarely been deliberate and this type of apparatus is therefore less easy to apply

(4) **Biopsy.** Careful bacteriological studies of the discharge, especially where purulent, must be carried out Actinomyces and tubercle bacilli may be hard to find and harder still to culture Repeated and deliberate search is, therefore, often necessary

(5) **Operative Measures.** Local operations on the track of an intestinal fistula are doomed to failure, where an incomplete diagnosis has been made Suspicion of the presence of a foreign body and its discovery forms the exception Wide opening up of fistulous tracks and the use of antibiotics of proved value may help but it is most important that sensitivity tests should be done and antibiotic therapy not employed at random The local application of X-rays has also been helpful in occasional cases

Resection and exclusion operations, however, hold pride of place, the latter being preferably of the end-to-side and not the side-to-side variety This is particularly true in regional ileitis and in the management of fistulæ in the region of the ileo-cæcal angle

Proximal diversion of the fæcal stream as a temporary or permanent step may be necessary in dealing with some fistulæ of the colon Where total colectomy is required, the proximal end of the defunctioned portion is better brought out as a mucous fistula (Fig. 3) between stages, than inverted and returned to the peritoneum. Irrigation of the lower segment is thus possible and the risk of a "blow out" much diminished. Finally, emphasis must be laid on the importance of wide resection and adequate drainage where an intestinal fistula has been of long standing and particularly where more than one track has been present.

cream (especially No. 71) and specific ointments, such as dilute (1 per cent) hydrochloric acid ointment for a high small intestine fistula, all have their uses but from time to time will fail

A dry skin with as little pressure as possible on it is the ideal to be aimed at, and



FIG 4 Large intestine mucous fistula during stage colectomy.

general measures such as liberal administration of vitamins, correction of anæmia and hypoproteinæmia, where present, are all important

(3) **Special Appliances.** The evolution of protective apparatus for intestinal fistulæ has made great strides since the wide adoption of ileostomy in the treatment of ulcerative colitis. Two main types have been evolved and personal preference must be expressed for the bag pattern as opposed to the cup and belt The bag, as in the Down Bros. pattern, may be of cellophane, being attached to an adhesive square, fitting over the fistula and sticky on both sides

The alternative, or Koenig-Rutzen type, depends on the principle of a cup, fitting over an exteriorized piece of bowel, preferably of the "spout" variety and perhaps

In thin subjects, especially females, with a narrow epigastric angle, the stomach is much more elongated and angulated, constituting *the J-shaped stomach*, whilst in broadly built men with a wide epigastrium there may be little gastric angulation at all—*the steer-horn stomach.*

Blood Supply of Stomach and Duodenum

The stomach has an abundant arterial supply derived from the right and left gastric and gastro-epiploic arteries forming arcades along the lesser and greater curves respectively, and from the short gastric arteries distributed to the upper part of the greater curve

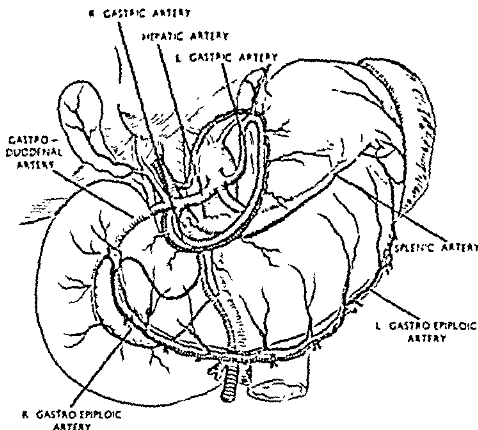


FIG. 6 The blood supply of the stomach.

and fundus (*see* Fig. 6). There is a very free anastomosis between the branches of these vessels in the wall of the stomach and this usually suffices to maintain the vitality of the organ after three or more of the major vessels have been ligated. The arterial supply to the duodenum comprises the supraduodenal branch or branches from the hepatic artery, many small twigs from the main stem of the gastroduodenal artery as it runs behind the first part of the duodenum, and the superior and inferior pancreatico-duodenal arteries coming from the gastroduodenal and superior mesenteric arteries respectively. The venous drainage from the stomach and duodenum is by veins which accompany the main arteries and enter the portal system.

Lymphatic Drainage of Stomach

This is of the utmost importance in the surgical treatment of gastric cancer and was early investigated by Jamieson and Dobson (1907) using the injection technique of

CHAPTER II

THE STOMACH AND DUODENUM

J. C. Goligher

SURGICAL ANATOMY AND PHYSIOLOGY

Subdivisions of Stomach

In the living subject, as contrasted with the fixed post-mortem body of the anatomy room, there are considerable variations in the shape and position of the stomach according to the amount of food it contains and to the position and bodily habitus of the individual. But the following parts can generally be recognized (*see* Fig. 5): *the cardia* where the œsophagus enters and where the circular musculature is thickened to form the *cardiac sphincter*, perhaps the most fixed point in the stomach; *the fundus*, lying above the level of the cardia; *the body*, or main part of the stomach cavity; *the pyloric portion*, the terminal more tubular segment of which is known as the *pyloric antrum*, the *pylorus*, which is the seat of *the pyloric sphincter*, demarcating the stomach from the first portion of the duodenum, or *duodenal bulb*. Connecting the cardia with the pylorus on the superior, concave, aspect of the stomach is the *lesser curve*, which has a descending portion, in relation to the body of the stomach, very liable to peptic ulceration, and an ascending portion in the pyloric antrum, more often attacked by carcinoma; the angle between these two parts of the lesser curve is known as the *incisura angularis*. Connecting the cardia and the pylorus on the convex aspect of the stomach is *the greater curve* which forms also the dome of the fundus. *The pyloro-duodenal junction* is indicated on the surface anteriorly by the presence of two veins running vertically upwards and downwards from the greater and lesser curves respectively and as a rule just failing to meet one another; these are the veins of W. J. Mayo, who first drew attention to their value as anatomical landmarks. The precise situation of the junction of the pylorus and duodenum can be further determined by noting on careful palpation the much greater thickness of the gastric than the duodenal wall.

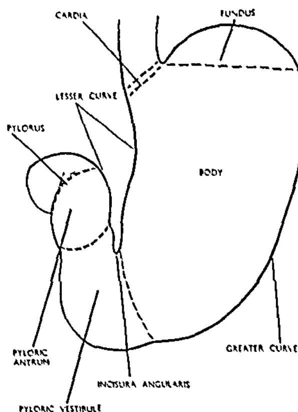


Fig. 5. Diagram indicating the parts of the stomach

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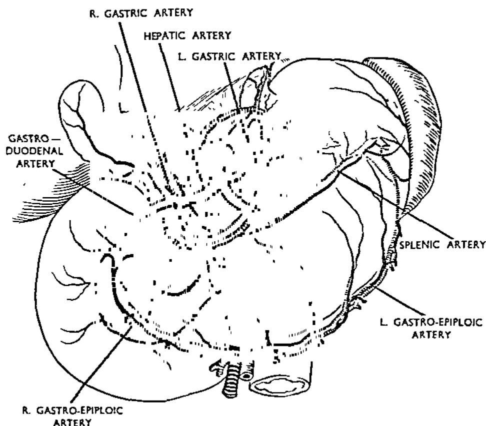


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Gerota. The intramural lymphatic plexuses in the wall of the stomach drain into the extramural lymphatic vessels and glands, which for the most part are disposed alongside the main arteries and veins. The lymphatic drainage of any part of the stomach is therefore primarily to the glands in relation to the main artery supplying that part and in accordance with the arrangement of the gastric arterial supply the stomach has been arbitrarily subdivided into three main lymphatic territories as in Fig. 7.

Zone I comprises the upper two-thirds of the organ and it drains into the glands lying on the arcade formed by the right and left gastric arteries along the lesser curve.

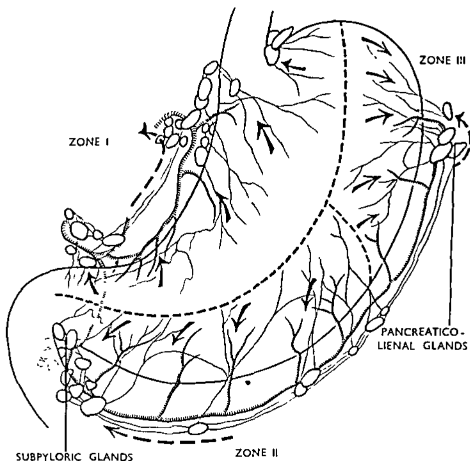


FIG 7 The lymphatic territories and drainage of the stomach Zone I includes the upper two-thirds of the stomach, Zone II the right two-thirds of the lower third, and Zone III the left third of the lower third

Outlying members of this glandular chain at its right end are the glands alongside the stem of the hepatic artery above the point where it gives off the right gastric artery. At the left end the "necklace" of glands around the cardia is to be noted. Ultimately all these glands on the lesser curve drain into glands on the main stems of the hepatic and left gastric arteries and around the cœliac artery from which these vessels spring.

Zone II includes the right two-thirds of the lower third of the stomach and drains into the glands on the right and left gastro-epiploic vessels. These glands are especially large and numerous just below the pylorus where they are known as the subpyloric glands. They drain mostly to the right and upwards along the gastro-duodenal artery to the hepatic and cœliac glands. Some of the glands further to the left may drain along the main stem of the left gastro-epiploic artery to glands on the gastro-splenic

and pancreatico-lienal ligaments near the hilum of the spleen and on the splenic artery as it is traced backwards to its origin from the cœliac artery.

Zone III represents the left one-third of the lower third of the stomach. It is drained by lymphatics which accompany the short gastric branches of the splenic artery to reach glands in the splenic hilum and on the trunk of the splenic artery along the upper border of the tail and body of the pancreas; ultimately the cœliac glands may be implicated.

It should be emphasized that the lymphatics in the gastric wall communicate freely, so that lymph—and carcinoma cells—from any of the "lymphatic areas" described above may not pursue the usual course of drainage but may pass to other glands, especially is this so if the glands which would ordinarily be the recipients are blocked with carcinomatous deposits as in an advanced growth.

Physiology of Stomach

The stomach acts as a reservoir for the food, which is intimately mixed by gastric peristalsis with the digestive juices secreted by the stomach and finally discharged in instalments through the pyloric sphincter into the duodenum It also exerts an important function in controlling the formation of red blood cells by the bone marrow.

Gastric Secretion. *The digestive secretion* of the stomach consists of hydrochloric acid and pepsin, which are produced by the oxyntic and pepsin-secreting cells of the gastric mucosa respectively. These are found *only in the gastric glands of the fundus and body of the stomach;* the glands of the pyloric region secrete merely alkaline mucus. Secretion of acid and pepsin is probably continuous in most people, but it is greatly augmented in response to the ingestion of food. The mechanism of this response is partly nervous and partly hormonal. *The nervous or psychical secretion* is due to nerve impulses generated in the brain by the sight, smell, and taste of food and mediated through the vagi to the stomach. *The hormonal secretion* depends on the production in the mucosa of the pyloric antrum, as a result of direct contact with the food, of the hormone "gastrin" which is conveyed by the blood stream to the glands of the body and fundus of the stomach and stimulates them to a further secretion of acid and possibly pepsin. Smaller quantities of a hormone having the same effect as gastrin are also produced by contact of food with the duodenal and jejunal mucosa, thus providing a subsidiary duodenal or intestinal phase of hormonal secretion. In the dog, as Dragstedt (1950) and others have shown the liberation of gastrin by the stomach itself takes place only in the pyloric antrum It is not known precisely how extensive is the area of production of gastrin in the human subject, but that the antrum is certainly capable of powerfully stimulating gastric secretion in man has been confirmed by several surgeons (McKittrick and Moore, 1945, Ogilvie, 1947).

The secretion of mucus by the stomach occurs from the glands of all parts of the mucosa as a result of direct contact with the food and drink, especially if these are chemically or mechanically irritating. The duodenal mucosa, from the pylorus to the opening of the common bile duct and pancreatic duct, contains Brunner's glands which secrete an even more alkaline type of mucus than does the gastric mucosa, but the exact mechanism governing the secretion is not understood.

Gastric Motility. This has been investigated by radiological examination after a meal containing an opaque medium. Peristaltic waves generally begin as slight contractions about the middle of the stomach and travel towards the pylorus, becoming

Gerota. The intramural lymphatic plexuses in the wall of the stomach drain into the extramural lymphatic vessels and glands, which for the most part are disposed alongside the main arteries and veins. The lymphatic drainage of any part of the stomach is therefore primarily to the glands in relation to the main artery supplying that part and in accordance with the arrangement of the gastric arterial supply the stomach has been arbitrarily subdivided into three main lymphatic territories as in Fig. 7.

Zone I comprises the upper two-thirds of the organ and it drains into the glands lying on the arcade formed by the right and left gastric arteries along the lesser curve.

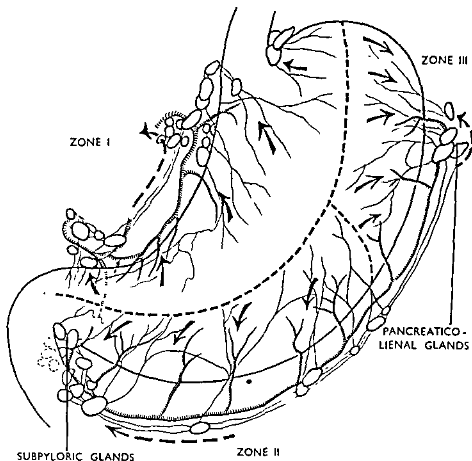


FIG 7 The lymphatic territories and drainage of the stomach Zone I includes the upper two-thirds of the stomach, Zone II the right two-thirds of the lower third, and Zone III the left third of the lower third

Outlying members of this glandular chain at its right end are the glands alongside the stem of the hepatic artery above the point where it gives off the right gastric artery. At the left end the "necklace" of glands around the cardia is to be noted. Ultimately all these glands on the lesser curve drain into glands on the main stems of the hepatic and left gastric arteries and around the cœliac artery from which these vessels spring.

Zone II includes the right two-thirds of the lower third of the stomach and drains into the glands on the right and left gastro-epiploic vessels. These glands are especially large and numerous just below the pylorus where they are known as the subpyloric glands. They drain mostly to the right and upwards along the gastro-duodenal artery to the hepatic and cœliac glands. Some of the glands further to the left may drain along the main stem of the left gastro-epiploic artery to glands on the gastro-splenic

However pain is sometimes experienced with gastric ulcer and gastric cancer when there is a complete achlorhydria, which is difficult to explain on the acid theory. It is known, moreover, that pain originating in the intestine is usually due to increased tension or spasm of the smooth muscle in its wall, and Hurst (1911) has claimed that a similar mechanism accounts for gastro-duodenal pain. According to his view the relief which undoubtedly attends the taking of alkalis in most ulcer cases is due to their abolition of spasm. In some patients suffering from peptic ulcer spasm of the pyloro-duodenal region is a notable feature on radiological examination, but in others with ulcers or carcinomata it is lacking. Furthermore the pain of a peptic ulcer is continuous and does not exhibit rhythmic variations like an intestinal colic. Enough has been said to indicate the unsettled state of opinion on the subject.

Hæmopoietic Function of Stomach. The work of Castle (1929) has shown that the proper maturation of the megaloblast precursors of normal erythrocytes depends on the absorption from the alimentary tract of a "hæmatinic principle." This is formed by the interaction of an "intrinsic factor," which is secreted by the mucosa of the body of the stomach, and an "extrinsic factor" which is contained in the food and is compounded of vitamin B12 and folic acid. Absence of the "hæmatinic principle" leads to the development of a macrocytic or pernicious type of anæmia. In ordinary pernicious anæmia the essential defect is believed to be a failure of production of the intrinsic factor due to the atrophic gastritis which is invariably present in these cases and which has also led to a complete achlorhydria. A similar interference with the formation of the intrinsic anti-anæmic factor could theoretically result from extensive surgical removal of the stomach, but after high subtotal gastric resection and even total gastrectomy a macrocytic anæmia is actually rare, though a microcytic iron deficiency anæmia is not at all uncommon (*see* page 194).

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

This is a disease of early infancy which affects boys at least four times as often as girls, and is especially prone to occur in first-born children. The muscle coat in the pyloric region undergoes a marked hypertrophy, which involves chiefly the circular muscle fibres and only slightly the longitudinal ones. This results in the formation of a firm rounded bobbin-like swelling of the consistency of cartilage, one inch or more in length, and of pale colour. The mucosa in the pyloric canal is thrown into deep folds by the narrowing of the lumen and these increase the obstruction further. Proximally the hypertrophy lessens gradually till the normal thickness of the gastric wall is reached, usually about the incisura angularis; distally the pylorus projects into the first part of the duodenum much as the cervix uteri does into the vagina, so that a circumferential "duodenal fornix" exists. The wall of the body and fundus of the stomach hypertrophies in an effort to overcome the obstruction, and eventually dilates; its mucosa may show a mild catarrh.

Several theories have been formulated to account for this condition. As the name implies it was originally believed that the disease represented a congenital hypertrophy of the pyloric muscle, the obstruction being due to the thickening of the muscle and to associated spasm. It is however doubtful if the condition is really congenital, because in most cases symptoms do not arise for some time after birth. The popular view is that of John Thompson (1921) who believed that the essential factor in the ætiology of the

gradually deeper till they end finally with a contraction of the pyloric sphincter itself. Two or more contractions may be in progress simultaneously at different stages. As digestion proceeds the waves may commence at progressively higher points on the body of the stomach till the whole organ is involved by them. Occasionally a different form of peristaltic activity seems to occur; a deep constriction ring is seen to develop at the junction of the body and the pyloric portion of the stomach, separating these two parts; the pyloric segment then undergoes a concentric contraction. The emptying of the stomach is regulated by the well defined pyloric sphincter, relaxation of which allows not only egress of acid gastric contents but also regurgitation of alkaline juices from the duodenum. The discharge of contents through the pylorus commences very soon after a meal, and the normal stomach should be completely empty within four or five hours. The influences controlling the delicate activities of the pyloric sphincter have not been satisfactorily elucidated.

The empty stomach is by no means inactive, for it frequently exhibits vigorous "hunger contractions" which may be responsible for the "pangs of hunger."

Nerve Supply of Stomach and Duodenum.

Motor and Secretory Innervation

The stomach and duodenum receive their sympathetic supply from the splanchnic nerves through the cœliac and superior mesenteric plexuses, and their parasympathetic supply from the vagi In most animals the sympathetic fibres are inhibitory to the main musculature of the stomach, and cause only a secretion of mucus, whilst the parasympathetic fibres are motor to the body of the stomach, inhibitory to the pyloric sphincter and produce an abundant secretion of gastric juice rich in hydrochloric acid and pepsin. In man it is to be noted that after surgical division of the splanchnic nerves—an operation which is occasionally used in the treatment of essential hypertension—there is little demonstrable effect on the motor or secretory activities of the stomach, but after complete vagotomy a pronounced atony of the stomach with severe retention often results, together with a profound depression of secretion. The effect of vagotomy on gastric motility gradually diminishes in time; there is some difference of opinion as to the permanence of its secretory effects.

Sensory Innervation

There are probably sensory fibres in both the vagi and the splanchnic nerves, but the fact that the former can be completely divided without affecting the appreciation of pain in the stomach or duodenum (Dragstedt, Woodward, Hooper and Storer, 1948), whilst division of the latter seems to block pain impulses from uncomplicated gastric or duodenal ulcers (Bingham, Ingelfinger, and Smithwick, 1950), suggests that the main sensory pathway is along the splanchnic route.

Mechanism of Gastric and Duodenal Pain. The actual stimulus that causes ulcer pain is not known. The more or less consistent association of acid with peptic ulcer and the fact that pain can be produced in patients with active ulcers by introduction of acid into the stomach, and relieved by withdrawal of the acid, and that it is not reproduced by re-introduction of the same volume of fluid after neutralization, has suggested to Palmer (1926) and Bonney and Pickering (1946) that acid is the effective stimulus.

of Ramstedt (1912). The principle of this procedure is the complete division of the circular pyloric muscle fibres by a longitudinal incision down to the mucosa (for details *see* page 205).

Post-operative feeding is commenced three hours after operation with a feed of 8 cc. of 5 per cent glucose in water every hour for six hours. Subsequently 30 cc. of peptonized milk is allowed two-hourly for the remainder of the first 24 hours. Next day the feeds are increased and given less frequently so that by the end of 48 hours from the time of operation the child may be put to the breast.

Results of Ramstedt's Operation. The results depend on the general nutritional condition of the infant at the time of operation and on the avoidance of *enteritis* which is responsible for most of the deaths after surgical treatment. The nursing of the babies in separate rooms with special nurses or at home after operation greatly reduces the risks of enteritis or of chest infections and is an important factor in achieving a low operative mortality. Many pædiatric surgeons have never had a death after operation for pyloric stenosis in private practice, where these conditions can be more easily obtained, but almost equally good results have been recorded in hospital practice from some centres.

HYPERTROPHIC PYLORIC STENOSIS IN ADULTS

Occasionally an hypertrophic pyloric stenosis is encountered in adult patients. Sometimes there is a history of symptoms dating from childhood suggesting that the condition may have been a persistence of an infantile stenosis, but in other cases the symptoms begin in adult life and may resemble those of a stenosing peptic ulcer or carcinoma. Radiological examination shows an obstructive pyloric lesion with a smooth conical termination to the gastric shadow, but it is usually impossible to diagnose this with confidence as not being due to a carcinoma. At operation the confusion in diagnosis often persists, so that whilst this lesion could theoretically be treated by Ramstedt's operation or by gastro-enterostomy, it is always safer to carry out a resection as for a pyloric carcinoma.

INJURIES OF THE STOMACH

Wounds of the Stomach

The stomach may be perforated or torn *from without* by stab- or missile-wounds, *from within* by swallowed foreign bodies with a sharp edge, including swords, or by an incautiously passed gastroscope or œsophagoscope. It should be borne in mind that with external injuries the wound of entrance does not necessarily lie in the epigastric region; it may be situated lower down in the abdomen, in the loin, or in the chest and the track proceed obliquely to the stomach. Also, other organs besides the stomach may be injured. The size of the wounds in the gastric walls—and usually the stomach is penetrated through and through—varies with the circumstances of the injury, but a direct antero-posterior wound with a high velocity bullet may produce very small perforations so that little gastric contents escape and spontaneous recovery may occasionally occur.

The symptoms and signs are essentially those of a perforated peptic ulcer, but as a rule it is quite impossible to be sure on clinical grounds that it is the stomach that is injured and not some other hollow viscus. Sometimes blood may be vomited. *Operation*

disease is a disturbance of the normal mechanism of neuromuscular co-ordination which controls the contraction and relaxation of this part of the alimentary tract. Pyloric inco-ordination is produced and this results in forcible and prolonged contraction of the pyloric muscle. Thompson postulated that the overaction of the muscle leads to its hypertrophy. The condition is in many ways similar to cardiospasm or achalasia of the œsophagus, but it is to be noted that in the latter disease no hypertrophy of the musculature of the obstructing part occurs.

Clinical Features

The child is well for the first week or two after birth, and then begins to vomit a few minutes after each feed. The vomited material consists of unaltered food with a little mucus but no bile. Little is passed per rectum and the child rapidly loses weight. Sometimes this constipation excites more attention than the vomiting. In other cases for some unknown reason diarrhœa occurs. When the condition has been present for two or three weeks visible gastric peristalsis may be observed or a palpable tumour may be detected in the pyloric region. These signs have often to be very carefully sought to be elicited. The child's abdomen should be observed after a small feed has been given. A swelling may then be seen to commence in the left subcostal region and to move across the epigastrium, and this phenomenon may be repeated many times. The point of termination of the waves of peristalsis indicates the probable site of the palpable pyloric mass.

In the later stages of the disease vomiting is almost continuous, the child becomes dehydrated and depleted in salt, and this is reflected in the sunken cheeks and anterior fontanelle, the dry tongue, scanty urine, and high blood urea. The diagnosis, however, should always be possible before this stage is reached. Sometimes help in recognizing the condition is obtained from radiological examination after instillation of lipiodol, or from the passage of a stomach tube four hours after a normal feed demonstrating a large gastric residue.

Differential Diagnosis

Simple conditions such as gastric catarrh and pyloro-spasm due to unsuitable diet and enteritis are excluded by noting that there is no retention of food in the stomach. *Congenital duodenal obstruction* is usually situated below the entrance of the common bile duct so that the vomitus contains bile; moreover the symptoms date from birth.

Treatment

Medical treatment, consisting of small frequent feeds with reduced fat content, gastric lavage before each feed, and the use of antispasmodic drugs notably eumydrine, may be tried for a few days If weight is gained and the amount of gastric residue diminishes, conservative measures should be continued. Otherwise operation should be performed. Babies who are still breast fed are probably best submitted to immediate operation, for medical treatment necessitates bottle feeding. Late cases with gross dehydration require intravenous or subcutaneous saline infusions for 24 or 36 hours before proceeding to operation. Otherwise the only preparation required is a stomach wash-out just before operation, to remove decomposing contents and air.

The operation which is now universally used for this condition is the pyloromyotomy

X-ray examinations of the abdomen are carried out to follow the progress of the body through the stomach and duodenum. If an irregular body is retained in the stomach or —even more serious—in the duodenum for more than a few days or a week it is probably better to remove it by operation. The extraction of a foreign body from the stomach is a simple manœuvre. Bodies impacted in the duodenum are usually better removed by manipulating them back into the stomach and incising the latter rather than the duodenum.

HAIRBALL-TRICHOBEZOAR AND PHYTOBEZOAR

Trichobezoar

Girls who are in the habit of biting their hair sometimes accumulate this in the stomach where the individual hairs are rolled together into a solid mass known as a hairball. Why the hair remains in the stomach in this way instead of being passed is not known, though there is some experimental evidence to suggest that a high fat diet may be important in the formation of a hairball. Examination of the mass shows that it consists of innumerable hairs mixed with all kinds of food material, especially fatty food. Sometimes the hairball forms a complete cast of the stomach with extensions into both the œsophageal and pyloric orifices. In some long-standing cases a chronic peptic ulcer has developed on the lesser curve.

A hairball may be symptomless or may give rise to vague epigastric pain and vomiting, the vomitus occasionally being bloodstained. Usually the mass can be palpated as a mobile rounded swelling not unlike a floating kidney. Radiologically the condition is easily recognized after an opaque meal as a central filling defect in the outline of the stomach.

The treatment consists of removal by gastrostomy, the abdomen being carefully explored to exclude other hairballs lower down in the gastro-intestinal tract. If an associated gastric ulcer should be present it will probably heal spontaneously. After this operation an effort should be made to avoid recurrence of the condition by stopping the habit of trichophagy, but this may be difficult—especially as some of these patients exhibit a psychopathic personality.

Phytobezoar

Sometimes a mass similar to a hairball is formed out of vegetable material such as coconut fibres and persimmons. It presents much the same clinical features as does a trichobezoar and is treated along identical lines.

VOLVULUS OF THE STOMACH

Complete volvulus consists of a rotation of the whole stomach either on its long axis—organo-axial—or on the axis of the lesser omentum—mesenterio-axial. In the former type the greater curve usually passes upwards forwards and to the right, carrying the greater omentum and sometimes the transverse colon with it; rarely is the rotation in the reverse direction. Usually no cause for the volvulus can be discovered but in some cases imperfect descent of the diaphragm leading to an abnormal roominess in the upper anterior part of the abdomen may be the explanation. Mesenterio-axial volvulus can only occur if the duodenum and pylorus are unduly mobile.

is urgently required as for any other perforating wound of the abdomen. Access to the abdomen is usually best gained by a right paramedian incision though sometimes when the wound traverses the lower chest a better route may be along the line of entry, i.e. a thoraco-abdominal approach. The extent of the abdominal injuries is determined and the opposing holes in the stomach wall are closed by suture. *Post-operatively* parenteral fluids and antibiotics are administered and gastric suction is instituted via an indwelling Ryle's tube for a few days.

Subcutaneous Rupture of the Stomach

Rupture of the stomach may result from blows on the epigastric region or crushes of the upper abdomen, especially when gastric distension is present. It has also been produced by blast in water though this more usually causes intestinal injury. It is said that the commonest site of gastric injury is on the anterior wall near the pylorus. There may be associated ruptures of other organs. *The clinical features and treatment* are as for a perforated peptic ulcer.

FOREIGN BODIES IN THE STOMACH AND DUODENUM

The rule has frequently been stated that objects capable of passing down the œsophagus can be relied upon to negotiate the pylorus if given sufficient time. This certainly holds for adults and for the majority of smooth objects liable to be swallowed, such as coins, buttons, and fruit stones. In young children however the pylorus is narrower than the narrowest part of the œsophagus so that a fairly large swallowed foreign body may be retained on that account alone. Also, in both adults and children irregular objects such as dental plates, forks, etc., may become impacted in the pyloric antrum or caught in the folds of the gastric mucous membrane. Another reason why metallic foreign bodies, like nails and pins may be retained in the stomach is that their combined weight may cause the stomach to sag into the pelvis, making their ascent out of the gastric sump to the pylorus progressively more difficult. The same gravitational mechanism may later cause delay in transit through the third part of the duodenum. Most foreign bodies in the stomach are swallowed accidentally in the food or in other ways, for example children playing with coins or kirby grips sometimes swallow them, and seamstresses and tailors may inadvertently ingest pins held between the lips. The most formidable collection of ironmongery such as knives, spoons, forks, and even lavatory chains, is occasionally swallowed by inmates of mental hospitals!

Treatment

Most smooth foreign bodies will pass spontaneously, though they may remain in the stomach for several weeks. There should therefore be no hurry to remove them by laparotomy. But if retained for a long time they may lead to ulceration or perforation and therefore should not be left indefinitely.

Irregular foreign bodies are liable to perforate the stomach or duodenum. Their passage may be facilitated and made safer by administering cotton wool or chopped up worsted in porridge or milk pudding. As a result of the churning activities of the stomach the wool becomes wrapped round the body padding its points. Aperients are to be avoided as the vigorous peristalsis they produce might increase the risk of perforation. The stools should be strained through muslin to recover the body, and periodical plain

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muscles and slight distension of the jejunum and ileum during the post-operative period caused the entire small gut to sag, dragging on its mesentery and stretching the superior mesenteric vessels tightly across the duodenum with resulting constriction and partial obstruction. However in only a proportion of the cases does the distal limit of the dilatation correspond with the point of crossing of the duodenum by these vessels. Further, the condition has occurred after gastro-enterostomy which would short-circuit any such obstruction. Another view is that the condition is due to excessive secretion of gastric juice in response to the action of a secretagoguic toxin. If this were true the manner of production of the secretagogue would then have to be explained. That it is not the true explanation however is shown by the fact that in most cases the dilatation, at any rate in the early stages, is due entirely to distension with air, and when it has been overcome by the passage of a stomach tube little fluid is withdrawn subsequently. The most popular view at the moment attributes the condition to a combination of ærophagy and paralysis of the stomach wall. Air swallowing is not uncommon during and after abdominal operations as McIver (1927) has shown. In the ordinary way the gastric distension which is produced is relieved by forcible expulsion of the air along the œsophagus. If for some reason not understood there is a paralysis of the stomach wall or a spasm of the cardia this natural relief is denied the patient and progressive gastric dilatation may occur.

Clinical Features

Rapid distension of the abdomen occurs and there is a certain amount of epigastric tenderness with a marked tympanitic note on percussion of this region and the lower chest. Vomiting may be absent at first but occurs later and the vomitus consists of mucus, bile, and possibly altered blood. The condition is associated with great initial collapse, the pulse being rapid, the temperature subnormal. Later if vomiting becomes more profuse the ill effects of dehydration and salt depletion are added. Though gravely ill the patient usually remains remarkably clear mentally. The decline in his condition if untreated is usually very rapid, often in a few hours.

Diagnosis

The condition most likely to be confused with acute dilatation of the stomach is paralytic ileus, but this is more insidious in onset and pursues a less dramatic course. Radiological examination of the abdomen is extremely helpful, as it shows the enormous gaseous distension of the stomach in acute gastric dilatation and the presence of gas and fluid levels in the small intestine in paralytic ileus.

Treatment

Acute dilatation of the stomach may perhaps be prevented by the early use of gastric aspiration via an indwelling Ryle's tube for post-operative abdominal distension or vomiting, or by the routine employment of post-operative gastroduodenal suction, as is the practice with many surgeons. Once the condition has developed however it is essential to pass a large bore stomach tube to secure rapid emptying of the stomach by suction and lavage. Thereafter a Ryle's tube may be substituted and constant suction maintained. The amount of fluid and electrolytes lost from the stomach should be replaced by an appropriate intravenous infusion, and the regime of suction and

Partial volvulus has been encountered as a complication of hour-glass contraction of the stomach due to gastric ulcer, or has been associated with upper abdominal adhesions or a diaphragmatic hernia.

Clinical Features

The symptoms produced are recurrent attacks of severe upper abdominal pain and distension, associated with outpouring of gastric juice in considerable amount into the twisted stomach. The patient may become very dehydrated. After a few hours or a day or so gas may be eructated and the symptoms may then completely subside. Radiological examination after a barium meal between attacks may reveal an appearance very like that of an hour-glass stomach.

Treatment

The passage of a large stomach tube may sometimes abort an attack. If attacks frequently recur and are severe laparotomy may be performed to fix the stomach and avoid further volvulus. This can be done either by carrying out a partial gastrectomy, preferably of Billroth I type, or by establishing a temporary gastrostomy. If the stomach is the seat of an hour-glass contraction gastrectomy will certainly be required.

ACUTE DILATATION OF THE STOMACH

This is a rare condition in which the stomach undergoes a rapid distension with gas and fluid so that it may fill the greater part of the abdomen. It occurs under the following circumstances: (*a*) Usually it follows an abdominal operation, most commonly one for a gynæcological or biliary lesion, exceptionally has it followed an operation on the stomach itself. (*b*) In some cases acute dilatation has occurred after simple manipulations such as the application or removal of a plaster jacket, or the performance of cystoscopy or urethral catheterization. (*c*) It has also been noted as an occasional complication after childbirth or various injuries. (*d*) It has been known to arise quite spontaneously in patients suffering from some form of wasting disease or disease of the spine. (*e*) Extremely rarely has acute dilatation occurred in otherwise healthy individuals.

The usual time for post-operative dilatation of the stomach to manifest itself is the second or third day after operation, but it has sometimes occurred earlier, and in a few cases has actually developed during the course of the operation. In these latter cases the stomach has rapidly become distended and within a few minutes has reached an enormous size, extending down into the pelvis and protruding through the wound. Passage of a stomach tube has resulted in the escape of a large quantity of gas of the same composition as atmospheric air, and the stomach has immediately resumed its normal size.

In established acute dilatation the stomach wall is stretched and thinned, its mucosa becomes fissured, and there is an exudate of offensive bloodstained fluid into the gastric lumen. In some cases the dilatation has been confined to the stomach; more usually it extends into the second and third parts of the duodenum or even into the upper jejunum. In roughly one quarter of the cases the dilatation stops short at the crossing of the third part of the duodenum by the superior mesenteric vessels.

There has been much speculation as to the cause of the condition but the precise mechanism of production is still obscure. One theory is that relaxation of the abdominal

muscles and slight distension of the jejunum and ileum during the post-operative period caused the entire small gut to sag, dragging on its mesentery and stretching the superior mesenteric vessels tightly across the duodenum with resulting constriction and partial obstruction. However in only a proportion of the cases does the distal limit of the dilatation correspond with the point of crossing of the duodenum by these vessels. Further, the condition has occurred after gastro-enterostomy which would short-circuit any such obstruction. Another view is that the condition is due to excessive secretion of gastric juice in response to the action of a secretagoguic toxin. If this were true the manner of production of the secretagogue would then have to be explained. That it is not the true explanation however is shown by the fact that in most cases the dilatation, at any rate in the early stages, is due entirely to distension with air, and when it has been overcome by the passage of a stomach tube little fluid is withdrawn subsequently. The most popular view at the moment attributes the condition to a combination of ærophagy and paralysis of the stomach wall. Air swallowing is not uncommon during and after abdominal operations as McIver (1927) has shown. In the ordinary way the gastric distension which is produced is relieved by forcible expulsion of the air along the œsophagus. If for some reason not understood there is a paralysis of the stomach wall or a spasm of the cardia this natural relief is denied the patient and progressive gastric dilatation may occur.

Clinical Features

Rapid distension of the abdomen occurs and there is a certain amount of epigastric tenderness with a marked tympanitic note on percussion of this region and the lower chest. Vomiting may be absent at first but occurs later and the vomitus consists of mucus, bile, and possibly altered blood. The condition is associated with great initial collapse, the pulse being rapid, the temperature subnormal. Later if vomiting becomes more profuse the ill effects of dehydration and salt depletion are added. Though gravely ill the patient usually remains remarkably clear mentally. The decline in his condition if untreated is usually very rapid, often in a few hours.

Diagnosis

The condition most likely to be confused with acute dilatation of the stomach is paralytic ileus, but this is more insidious in onset and pursues a less dramatic course. Radiological examination of the abdomen is extremely helpful, as it shows the enormous gaseous distension of the stomach in acute gastric dilatation and the presence of gas and fluid levels in the small intestine in paralytic ileus.

Treatment

Acute dilatation of the stomach may perhaps be prevented by the early use of gastric aspiration via an indwelling Ryle's tube for post-operative abdominal distension or vomiting, or by the routine employment of post-operative gastroduodenal suction, as is the practice with many surgeons. Once the condition has developed however it is essential to pass a large bore stomach tube to secure rapid emptying of the stomach by suction and lavage. Thereafter a Ryle's tube may be substituted and constant suction maintained. The amount of fluid and electrolytes lost from the stomach should be replaced by an appropriate intravenous infusion, and the regime of suction and

intra-venous drip should be continued for a day or two till it is clear that there is going to be no recurrence of the condition.

The method formerly advised of turning these patients into the prone position in the hope of relieving compression of the third part of the duodenum by the superior mesenteric vessels was based on an erroneous conception of the cause of the condition and was moreover extremely difficult to maintain because of the discomfort it caused to a post-operative patient.

GASTRITIS

Acute superficial gastritis may sometimes enter into the differential diagnosis of an acute upper abdominal condition such as a perforated peptic ulcer or an acute cholecystitis, demanding urgent surgical treatment. Similarly chronic gastritis may require to be distinguished from a chronic peptic ulcer or gastric carcinoma as a cause of dyspepsia. But the only form of gastritis that calls for active surgical treatment is the very rare *acute phlegmonous gastritis.*

This is an acute infective condition of the stomach wall which becomes enormously thickened, especially in its submucous layer. Eventually the serosa becomes involved and is covered with a fibrous exudate. Suppuration may occur in the gastric wall, leading to abscesses which may burst into the lumen or the peritoneal cavity. The whole stomach may be involved, or, more commonly, the process is confined to a part of the gastric wall. The disease may develop from a local lesion such as a peptic ulcer or gastric cancer, or may be a metastatic complication of a septicæmic condition such as puerperal fever or osteomyelitis. The appearance of the stomach in phlegmonous gastritis is said to be not unlike that of the small intestine in acute forms of Crohn's nonspecific enteritis.

Clinical Features and Treatment

The disease is of sudden onset and causes severe epigastric pain and tenderness, repeated vomiting and profound constitutional disturbance. Sometimes a palpable swelling may be detected. Accurate clinical diagnosis is extremely difficult, and if the patient is subjected to operation it may be equally difficult to decide on the correct procedure. The condition is probably best treated by resection, especially as there may be confusion with a carcinoma or the phlegmon may have arisen as a complication of a growth, but this may involve a total gastrectomy. Alternatively local drainage may be instituted.

TUBERCULOSIS OF THE STOMACH

Tuberculosis of the stomach is a rarity, which is remarkable when it is recalled that tubercle bacilli are not infrequently swallowed in the sputum by sufferers from pulmonary tuberculosis, and that they may survive for several hours in the gastric juice. Most examples of gastric tuberculosis occur in association with tuberculous lesions of the lungs, though occasionally the stomach wall may be infected from an adherent caseous gland.

The usual lesion found is a solitary ulcer on the lesser curve near to the pylorus. This exhibits the features of a typical tuberculous ulcer. Sometimes several small miliary ulcers are present. A hyperplastic form of the disease comparable to that which occurs in the ileocæcal region has also been described, it may closely resemble a carcinoma.

SYPHILIS OF THE STOMACH

This is also excessively rare. It commences as a gummatous infiltration of the submucosa, usually near the lesser curve and the pylorus. This eventually produces great thickening of the stomach wall and a polypoid mass which projects into the lumen. Central necrosis often occurs leading to the formation of a large ulcer with irregular edges and a sloughing base. Septic absorption takes place from the lesion and hæmorrhage may occur, these two events contributing to produce in the patient a distinctly cachectic appearance.

Clinical Features and Treatment

The condition may cause vague dyspeptic symptoms. A mass may be palpable in the epigastric region. Radiological examination usually shows an appearance not unlike that of carcinoma of the stomach, and gastric analysis often strengthens the suspicion of malignancy by showing complete achlorhydria. Under the circumstances a diagnosis of malignant disease is usually made and gastrectomy is performed, the demonstration of the true nature of the condition depending on histological examination. If other syphilitic manifestations arouse the clinical suspicion of a gumma of the stomach and this is strengthened by a positive Wassermann reaction, a short course of intensive antisyphilitic treatment may confirm the diagnosis by producing a marked retrogression of the lesion.

LYMPHADENOMA OF THE STOMACH

Sometimes Hodgkin's disease gives rise to a mass in the stomach wall which has the macroscopic appearance of a sarcoma or carcinoma. The condition is usually diagnosed and treated as a tumour and the true pathology is only revealed on subsequent histological examination.

PEPTIC ULCER

Since the beginning of the present century, when it was a barely recognized disease, peptic ulcer has become progressively more common, especially in the last decade, so that it is now one of the major maladies of mankind. It affects predominantly the peoples of Western European countries, and North America, is less common in Australasia and South America, and is rare in Africa or Asia (with the notable exception of Southern India) apart from European residents. It has been estimated by Doll and Avery Jones (1951) that the total number of persons in England and Wales who have, or have had, peptic ulcers may be as high as 1,449,000; that in any one year as many as 638,000 men may suffer from ulcer symptoms; and that peptic ulcer cases constitute about 10 per cent of all the inpatients and new outpatients at general hospitals!

Pathology

Peptic ulceration is believed to be due to digestion by the gastric juice and it may occur in any portion of the alimentary tract exposed to the action of this secretion. The stomach and duodenum are the common sites, but peptic ulcers may form also in the jejunum after the operations of gastroenterostomy or partial gastrectomy, in the œsophagus in cases where abnormal regurgitation of gastric contents occurs through the cardia, and in the small intestine in association with heterotopia of gastric mucosa in a

Meckel's diverticulum. Only gastric, duodenal, and jejunal ulcers will be considered in this chapter.

Acute Peptic Ulcers. Acute peptic ulcers occur as single, or more commonly multiple, small erosions of the mucosa *of any part of the stomach or upper duodenum.* They arise most frequently in the terminal phases of toxic and infective processes such as uræmia, tuberculosis, peritonitis, tonsillitis, and other septic conditions. The rather more extensive ulceration in the duodenum described by Curling in association with burns is to be regarded as being of this nature. Acute ulcers, single or multiple, may also appear occasionally in otherwise healthy people for no apparent reason.

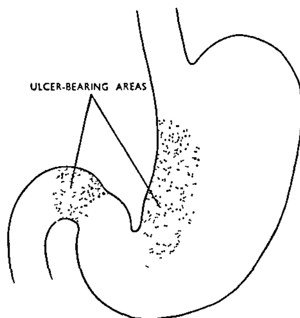


FIG 8 The distribution of peptic ulcers in the stomach and duodenum. Stippling indicates the "ulcer bearing areas"

Acute peptic ulcers are usually small rounded erosions which extend only into the submucosa; they may however enlarge to a size of one inch or more. Though usually superficial, they may result in erosion of a vessel and give rise to hæmorrhage, or they may deepen through all coats to perforate into the peritoneal cavity. Histological examination usually shows remarkably little inflammatory or fibrotic change in the surrounding gastric or duodenal wall. Probably most acute ulcers heal rapidly, but a few in certain areas of the stomach persist and give rise to chronic peptic ulcers.

Chronic Peptic Ulcers unlike acute ulcers, are restricted in their distribution with remarkable constancy to a small area of the stomach and duodenum. *This "ulcer-bearing area"* includes the lesser curve and adjacent anterior and posterior walls of the stomach from the œsophagus to a point one and a half inches from the pyloro-duodenal junction, and the entire circumference of the first inch or so of the duodenum (*see* Fig. 8). Some 95 per cent of all chronic ulcers occur within these bounds. Most ulcers apparently located at the pylorus or in the pre-pyloric region turn out in fact to be duodenal ulcers or pyloric carcinomata. An ulcerating lesion of the greater curve, fundus or cardia is also more likely to be malignant. Simple chronic duodenal ulcers on the other hand do occasionally occur in the second part of the duodenum. Sometimes two or more chronic ulcers are present in the one case—"kissing ulcers" of the anterior and posterior duodenal walls are not uncommon, and a coincidental gastric ulcer is found once in every thirty or forty cases of duodenal ulcer.

Duodenal ulcers are usually rounded and quite small, seldom being more than three quarters of an inch across and often much less. Gastric ulcers by contrast vary greatly in size but quite commonly reach a diameter of one inch or more; large ones tend to assume a saddle shape as they spread from the lesser curve on to the anterior and posterior walls. A chronic ulcer has a "punched out" appearance with clear-cut edges, and the surrounding mucosa may be thrown into radiating folds, particularly with

gastric ulcers (*see* Fig. 9). Almost invariably the ulcerative process has extended right through the muscular coat to the subserosa which is œdematous, fibrosed and thickened. The external appearance of the ulcer is especially important to the operating surgeon because with a small lesion there may be little to feel. The serosa overlying the ulcer is whitened due to the subserous fibrosis and a characteristic stippling is produced by irregularly arranged rather dilated small vessels. These changes are most easily detected with anterior wall duodenal ulcers; with lesser curve ulcers the attachment of the lesser omentum may mask the appearances. At a later stage the external signs may be more obvious in the form of gross puckering and scarring with adhesions to other organs such as the liver, gall bladder or pancreas. Posterior wall ulcers are especially liable to become closely adherent to the neck and body of the pancreas. If the ulceration deepens further, as it often does, it destroys the remnants of gastric or duodenal wall leaving fibrosed pancreatic or hepatic tissue in the floor of the ulcer—*the penetrating ulcer*.



FIG 9 A chronic peptic ulcer of the stomach. Note the "punched out" appearance, with clear-cut edges and radiating mucosal folds

MICROSCOPICAL FEATURES

Histological examination usually shows that the muscle coat of the gastric wall has ✓been completely destroyed in the base of the ulcer and replaced by fibrous tissue; a ✓fibrous inflammatory reaction is also evident in the margins of the ulcer. The arteries in the vicinity show a considerable degree of endarteritis obliterans which is perhaps beneficent in diminishing the risk of hæmorrhage from these vessels should they subsequently become eroded. But the combination of extensive fibrosis and endarteritis provides a poor basis for sound and permanent healing. At the edge of the ulcer peculiar distortions and displacements of the epithelium often occur simulating the appearance of a carcinoma, and in the past these irregularities have led some pathologists to erroneous conceptions of the frequency of malignant degeneration in simple peptic ulcers of the stomach. Most authorities in this country now rate the incidence of carcinomatous change in apparently simple gastric ulcers subjected to surgical excision as not more than 6 per cent (*see* page 45).

Ætiology

An enormous amount of information, derived from experimental and clinical sources, is now available on this controversial subject, but in this brief section only those data which seem to be particularly relevant to the problem of peptic ulcer in man will be considered. More comprehensive surveys are provided by Mann (1951) and Grossman (1951).

The Acid Factor. One of the mysteries of physiology is why the stomach is not ordinarily digested by the hydrochloric acid and pepsin which it secretes. Such digestion of the gastric mucosa is quite common as a post-mortem phenomenon, and has been

centres to produce severe gastric hypersecretion is well shown by the secretory response of the stomach to severe hypoglycæmia produced by an injection of insulin, a reaction which occurs only if the vagal pathway from the brain to the stomach is intact. Practical use is made of this reaction in the Hollander insulin test to determine post-operatively the completeness of a vagotomy (*see* page 71).

The Ulcer Diathesis. Hurst (1924) has emphasized that there is a predisposition or diathesis on the part of certain individuals towards peptic ulceration. This is most evident in regard to duodenal ulcer, which occurs characteristically in hyperasthenic, over-anxious, sensitive, active men who have usually a short steer-horn type of stomach which empties rapidly and produces a high level of secretion and acidity. The "gastric ulcer diathesis" is less clearly defined but is typified by the hyposthenic, visceroptrophic, listless patient with a long J-shaped stomach and rather low acid secretion. Whilst Hurst's contentions in regard to diathesis are in the main correct, gastric and duodenal ulcers are often encountered in atypical individuals.

Other Predisposing Factors. *Excessive smoking* has frequently been blamed for producing gastric hypersecretion and thereby predisposing to peptic ulceration. It is an undoubted fact that many ulcer patients are heavy smokers, but many others are non-smokers, and despite the great increase in cigarette smoking amongst women in the last decade there has been no heavier incidence of duodenal ulcer in female patients during that period. *Irregular and inadequate feeding* has also been incriminated as a factor in the development of ulcer. Obviously the leaving of a hyperchlorhydric gastric juice unbuffered by food for long periods may be detrimental in these patients. A deficiency of Vitamin C also may predispose to gastric ulceration in humans as it can be shown to do in experimental animals. It is highly likely however that hasty and irregular dietetic habits, like excessive smoking, are significant more as indications of the psychological type of individual than as direct casual agents of peptic ulceration. *Seasonal influence:* The tendency of ulcer symptoms to recur in winter and to be relieved in the summer or by a holiday probably reflect minor variations in general health which these seasonal changes are liable to produce. *Racial and geographical influence:* The well known variations in the incidence of peptic ulcer in different parts of the world have not been satisfactorily explained, but probably reflect in part differences in the mode of life in different countries, Western civilization being particularly ulcerogenic.

Factors Determining the Site of Ulceration. The strict localization of chronic peptic ulcers to the "ulcer bearing area" of the stomach and duodenum has evoked much speculation, but no very satisfactory explanation has been forthcoming.

The Theory of Localized Ischæmia

Wilkie (1911) drew attention to the fact that the anterior aspect of the first inch of the duodenum was supplied by an inconstant end-artery, the supraduodenal branch of the hepatic or right gastric artery, and it has been postulated that blockage of this vessel by spasm or embolism might result in an area of ischæmia resulting in the formation of a duodenal ulcer. This theory is not very convincing because at the operation of gastroduodenal resection for ulcer it can usually be seen that quite apart from this supraduodenal artery the duodenum has a very abundant blood supply, and also because it leaves unexplained the localization of the more common posterior wall duodenal

ulcer. Efforts by Reeves (1920) to account for the occurrence of gastric ulcers on the lesser curve on the basis of localized ischæmia are equally unconvincing.

THE THEORY OF INFECTION OF LYMPH FOLLICLES

Lymph follicles are numerous in the wall of the stomach and duodenum but especially abound in the "ulcer bearing area." It had been suggested that a localized follicular abscess may be the starting point of an ulcer.

THE THEORY OF TRAUMA TO THE LESSER CURVE OR FIRST PART OF THE DUODENUM

The "ulcer bearing area" of the duodenum comprises that part which receives the full "blast" of the acid gastric contents as they are expelled from the stomach and which is therefore exposed to the maximum of mechanical and chemical trauma. The common ulcer site in the stomach is on the "Magenstrasse" along which, according to some authorities passes the main volume of food entering the stomach, so that it may conceivably be subjected to an undue amount of trauma and attrition.

Clinical Features

Pain is usually the most prominent symptom of a peptic ulcer and has several characteristic features which are of great diagnostic significance. Almost invariably the onset of the pain bears a constant relationship to the taking of food. With an ulcer high on the lesser curve of the stomach the pain comes on half to one hour after meals, or sooner, and usually persists for an hour or so, passing off of its own accord so that there is a pain-free interval before the next meal. Sometimes vomiting occurs; and the pain is relieved by it; or the patient may take an alkali or a glass of milk with immediate relief. When the ulcer lies nearer the pyloric region or in the duodenum the onset of the pain is usually deferred for two or three hours after a meal, and persists till the next meal unless an alkaline mixture or snack is taken in the interval. Night pain is especially common in duodenal ulcer and the patients not infrequently keep a glass of milk or some form of alkali by their bedside to allay it. Initially the pain in ulcer cases is situated in the epigastric region but with posterior wall ulcers penetrating into the pancreas pain is usually also experienced in the back. Another characteristic feature of ulcer pain is that there are usually periods of complete remission lasting from a few days to several months, when the patient has no discomfort of any kind. The return of the dyspepsia is often associated with business or domestic worries, ill-health due to other causes, or climatic conditions, pain being more frequent in winter months. In long-standing cases the intervals between attacks may become gradually less and less and eventually the pain may be continuous and unrelenting. In these cases also vomiting may become a disturbing symptom if stenosis occurs.

Patients with peptic ulcer generally have a good appetite. With gastric ulcer there may be some loss of weight—often amounting to severe emaciation with large ulcers—but with duodenal ulcer the weight is usually well maintained and may indeed be increased due to frequent snacks which the patients have been in the habit of taking to relieve their pain. With some patients there is no history to suggest the presence of a peptic ulcer till a perforation or hæmorrhage occurs—the so-called "silent ulcer."

Clinical examination of a patient with a peptic ulcer may be entirely negative, but



(Reproduced by courtesy of Dr E Rohan Williams)

FIG. 10 Radiograph after a barium meal showing large gastric ulcer niche on the lesser curve



(Reproduced by courtesy of Dr E Rohan Williams)

FIG 11. Radiograph after a barium meal showing gastric ulcer on the posterior wall of the stomach with well marked radiating folds of mucosa.



(Reproduced by courtesy of Dr E. Rohan Williams)

FIG 12 Radiograph after a barium meal in a case of duodenal ulcer, the duodenal cap being deformed and a small ulcer niche evident in it

occasionally there is tenderness and slight rigidity in the epigastrium—high up in the mid-epigastrium with gastric ulcer, lower down and more to the right with duodenal ulcer. Hyperæsthesia of the skin in the epigastric region may sometimes be demonstrated but it is not consistently present. When stenosis exists splashing may be elicited in the dilated stomach on shaking the patient.

Special Investigations

Radiological Examination

Radiological examination after an opaque meal is the most valuable single diagnostic procedure in these cases.

A chronic gastric ulcer can almost always be demonstrated as a niche on the lesser curve or posterior wall, sometimes with a corresponding notch on the greater curve

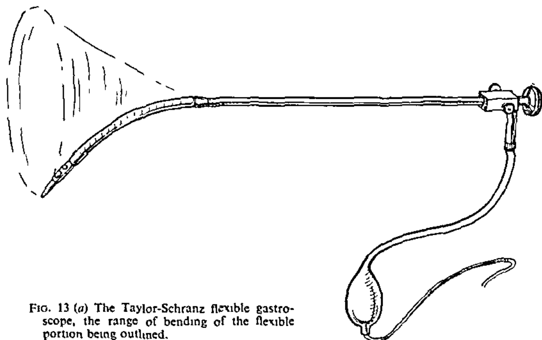


Fig. 13 (*a*) The Taylor-Schranz flexible gastroscope, the range of bending of the flexible portion being outlined.

opposite it due to spasm or fibrosis. There is usually tenderness over the crater and the mucosal folds can sometimes be seen to radiate from the lesion (*see* Figs. 10 and 11).

A duodenal ulcer crater is often more difficult to demonstrate but there are usually indirect signs in the form of tenderness and deformity of the duodenal cap and sometimes initial rapidity of emptying of the stomach with later delay. In some cases an ulcer niche may be revealed by a persistent fleck of barium (*see* Fig. 12). Radiological examination also shows whether there is any mechanical difficulty to the emptying of the stomach.

Gastroscopy

This is carried out by the semi-flexible gastroscope, of which the most commonly used pattern in this country is the Taylor-Schranz (1941) modification of the original German Wolf-Schindler instrument (*see* Fig. 13 (*a*)). It consists of a flexible lower third and a rigid upper two-thirds. At the extreme end of the flexible portion is a small metal component, about one inch long, containing the bulb for illumination and, just proximal to it, the objective. The flexible part itself is composed of two concentric steel spirals

containing a number of closely set biconvex lenses of short focal length and covered with two layers of rubber. Between these layers is an air passage, and the outer layer is perforated at its lower end to allow the air to find its way into the stomach during inflation.

The rigid portion consists of a long hollow steel tube containing a few lenses for the transmission of light, and an air passage. At the upper end of the rigid portion are the eye-piece, the attachment for the current and the bellows, and the wheels and axle for producing active flexion of the lower end. The range of active or passive flexion possible before the visual image becomes distorted is approximately 30 degrees.

The Technique of Gastroscopy. Examination is best performed early in the morning, no food or drink having been taken since 6.0 p.m. the previous evening. Heavy premedication is given with omnopon and scopolamine. Local anæsthesia usually suffices, the patient being given one or two gr. 1 tablets of decicain (anethocain) to suck for three-quarters of an hour before the instrument is passed; this may be reinforced by swabbing the oropharynx with decicain solution immediately before the endoscopy. In apprehensive subjects local anæsthesias should be supplemented by general anæsthesia using pentothal with one of the short-acting relaxant drugs.

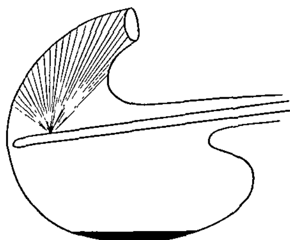


FIG 13 (*b*) Diagram showing gastroscope in position in stomach

The patient lies on his left side with his knees drawn up, and with his head blindfolded and resting on a small sandbag. An assistant controls the position of the head and the amount of extension of the neck. Holding the well lubricated gastroscope in his right hand rather like a large pen the surgeon introduces the flexible end through the right or left angle of the mouth on to the back of the tongue. Here its point is maintained accurately in the midline by the index and middle fingers of the left hand, and is forcibly depressed against the tongue so that it does not impinge on the posterior pharyngeal wall as it descends through the pharynx. When the cricopharyngeal sphincter is reached the conscious patient is asked to swallow to enable the instrument to be passed into the œsophagus; in the unconscious patient the sphincter is already relaxed. The subsequent course down the œsophagus into the stomach takes place very rapidly, the gastroscope being passed to its fullest extent in the first instance The current and bellows are then connected.

With only slight inflation of the stomach, the gastroscope is first of all turned to look upwards and slightly to the left in search of the pyloric antrum By active flexion of the point of the instrument in this same direction the pylorus itself can usually be seen (*see* Fig. 13 (*b*)). The stomach is then further inflated and the gastroscope is gradually withdrawn, being rotated repeatedly through 360 degrees to bring different parts of the stomach into view. Certain "blind areas" however exist for the gastroscope; these are the extreme upper end of the lesser curve, much of the posterior wall, the lesser curve of the pyloric antrum in markedly J-shaped stomachs, and the greater curve opposite the end of the instrument. Orientation in the stomach and accurate interpretation

of gastroscopic findings require considerable experience, without which gastroscopy may be valueless or misleading. The only real danger from gastroscopy is that of *perforation of the posterior wall of the pharynx or œsophagus in the immediate post-cricoid region.* This occurs once in every 500 to 1,000 gastroscopies and shows itself usually by causing surgical emphysema, and later cellulitis, in the neck and mediastinum. It may be treated by immediate suture or conservatively by stopping mouth feeding and giving massive doses of antibiotic (*see* Goligher 1948). Perforation of the stomach itself is extremely rare. For more detailed information regarding gastroscopy the reader is referred to Schindler (1937).

Gastroscopic examination is most usefully employed as a supplement to radiological investigation in cases of suspected peptic ulcer or carcinoma where the radiologist is unable to make up his mind as to the significance of the changes observed by him in the gastric wall, or where the radiological findings though definite run counter to clinical expectation. If the area under suspicion can be brought into view by the gastroscope it is often possible to confirm the diagnosis of a benign ulcer or malignant lesion, or to show that the gastric wall in that region is perfectly normal. The gastroscopic appearance of a benign ulcer and of a carcinomatous ulcer are usually quite distinctive.

The former is a clear cut circular or oval lesion as a rule with normal mucosa extending right up to the ulcer margin. There is no elevation of the edges. The floor of the ulcer is smoothly concave and covered with homogeneous grey slough (*see* Plate I, Fig. (*a*)). A malignant ulcer on the other hand is often more irregular in shape, it has raised nodular edges and the mucosa for some distance from the main ulcer may be seen to be infiltrated. The floor of the lesion is usually irregular, often nodular (*see* Plate I (*b*)). Unfortunately not all carcinomata are as distinctive as this and they may resemble to some extent a benign ulcer, particularly if they have arisen in a pre-existing ulcer. If the gastroscopist has any doubt as to the nature of an ulcerating lesion it should as a rule be regarded as malignant. But in elderly unfit subjects it may be justifiable to persist with medical treatment for two or three weeks and to review the ulcer with the gastroscope; sometimes the benign nature of the condition is then revealed by the evidence of healing and the general appearance of the lesion.

Gastric Analysis

Patients with duodenal ulcer generally show a high level of free HCl and total acid as estimated by the fractional test meal and by the histamine and insulin tests; also there is usually marked hypersecretion and hyperchlorhydria during the night. In gastric ulcer, however, normal or even subnormal findings are the rule. It is rare that gastric analysis is of much real value in diagnosis except in cases with gastric ulcers the benign nature of which may be in doubt; the finding of a complete achlorhydria, resistant to histamine stimulation, is suggestive that the lesion is a carcinoma. The discovery of a very high level of hyperchlorhydria and hypersecretion in cases of duodenal ulcer is of some prognostic significance and may have a bearing on the choice of operation.

The Presence of Occult Blood

The presence of occult blood in the stools when the patient is on a meat-free diet is corroborative evidence of the existence of an ulcer.

Diagnosis. In the diagnosis of a peptic ulcer the two most important considerations are the history and the radiological examination, and in most cases they suffice for a

juice to regurgitate through the stoma into the stomach and to neutralize to some extent the acidity of the gastric juice. By these two mechanisms most duodenal ulcers are caused to heal after posterior gastro-enterostomy. Unfortunately a certain percentage develop another ulcer in the jejunum adjacent to the stoma which causes more severe symptoms and more troublesome complications than the original duodenal lesion. Just how frequently stomal ulceration occurs after gastro-enterostomy is much debated. The estimates of different authorities have varied from 2–30 per cent or more (Walton, 1934, Wright, 1935, and Lowdon, 1948), and it is this variation that has resulted in gastro-enterostomy being both one of the most lauded and most condemned operations in surgery.

Undoubtedly some of the lower estimates have been due to an insufficiently long follow-up of patients, for stomal ulcer after gastro-enterostomy quite frequently does *not develop for many years after operation; unless the cases are traced for at least 5–10* years many instances of recurrent ulcer will be overlooked. Another factor accounting for discrepancies in estimates of the incidence of stomal ulcer is the difference in the selection of cases for operation by different surgeons. The patients who are most likely to develop jejunal ulcers are the young or middle-aged individuals with high gastric acidity. After gastro-enterostomy their hyperchlorhydria often continues little abated and constitutes a serious threat to the relatively susceptible jejunal mucosa exposed at the stoma. If the operation is reserved for older patients with lower acidity, and particularly if their lesions have caused some duodenal stenosis, the incidence of jejunal ulceration is found to be much less though not completely eliminated. Comprehensive surveys of hospital statistics (as contrasted with the more partisan and less reliable reports of individual surgeons), such as those of Pyrah (1935) and of Cooper (1949), show that some 80 per cent of patients submitted to gastro-enterostomy for duodenal ulcer obtain good results. Of the other 20 per cent with further symptoms, 10 per cent come to a second operation for stomal ulcer, and 10 per cent, though having intermittent dyspepsia, are able to carry on—some of them with relatively little discomfort and reasonably satisfied with their results. The immediate mortality of gastro-enterostomy should be very low—not more than 1 per cent.

Dissatisfaction with the high incidence of jejunal ulcer after gastro-enterostomy has led most surgeons to abandon this operation as a routine procedure in the treatment of duodenal ulcer, though many have retained it as an occasional operation for poor risk elderly patients, especially those with stenosing ulcers. For the average case they have preferred to employ an extensive partial gastrectomy. However it has been argued by Walton (1930), who was one of the leading advocates of gastro-enterostomy, that it is more reasonable to employ this relatively safe operation as the routine procedure, and to reserve partial gastrectomy with its greater risks for the cases that develop stomal ulcer after gastro-enterostomy. Now that vagotomy has been introduced this argument is being advanced in support of the combination of gastro-enterostomy and vagotomy *vis-à-vis* gastrectomy.

PARTIAL GASTRECTOMY. The performance of an extensive partial gastrectomy for duodenal ulcer was first suggested by Finsterer of Vienna (1918) and von Haberer of Cologne (1920), with the object of producing a hypo- or achlorhydria and thereby avoiding stomal ulceration. The lowering of gastric acidity by this procedure is achieved in two ways. Firstly, removal of the mucosa of the pyloric antrum eliminates the main

site of production of gastrin; and secondly, the proximal extent of the gastrectomy results in excision of a considerable part of the actual acid-secreting mucosa of the stomach. As usually performed the operation consists of resection of the distal two-thirds or more of the stomach and the first inch of duodenum containing the ulcer, and is completed by closure of the duodenal stump and anastomosis of the stomach remnant to the upper jejunum (Billroth II or Polya operation). Alternatively continuity may be re-established after resection by direct anastomosis of the gastric and duodenal stumps according to the Billroth I technique, but opinion is divided as to whether this procedure gives as good results in duodenal ulcer cases as does the Billroth II operation; it is to be noted particularly that von Haberer (1947) after an experience of several thousands of the former operations in the treatment of duodenal ulcers eventually abandoned this technique in favour of a high Billroth II type of resection. The present author has likewise had a higher incidence of recurrences after Billroth I than Billroth II resections of comparable extent in duodenal ulcer cases. The experience of Perman (*see* Sjögren, 1952) of frequent further ulceration after conversion of Polya gastrectomy performed for duodenal ulcer to the Billroth I type is also significant (*see* page 85). (For full details of these operative procedures *see* page 97.)

In the early days of gastrectomy the risks were much greater than those of gastro-enterostomy, but as surgeons have acquired more familiarity with the technical difficulties the hazards have been steadily reduced, and some very low mortality figures have been reported by acknowledged experts such as Pannett (1938) (0·86 per cent), Hosford (1949) (0·5 per cent), and Tanner (1951) (1·5 per cent). The average mortality in the hands of experienced gastric surgeons is probably now about 3 per cent, and this is the figure that has been reported fairly consistently from the Mayo Clinic over the past few years for gastrectomy for duodenal ulcer where some 400 of these operations are performed annually.

As regards freedom from jejunal ulceration, the fact must be faced that gastrectomy has been somewhat disappointing; stomal ulcers do occasionally occur, particularly if the resection has been limited in its extent. This has led some surgeons to employ more and more extensive gastrectomies involving removal of at least three-quarters or four-fifths of the stomach, sometimes leaving the merest gastric remnant nourished by one vas brevis, as recommended by Visick (1948). Most surgeons however have been content to practise a two-thirds or three-quarters gastrectomy. With a modified Polya resection of this extent Tanner (1951) has reported a 2–3 per cent incidence of stomal ulceration in a 5 year follow-up. Maingot (1953) and the writer have had a similar frequency of recurrence after a rather more extensive gastrectomy also of Polya type.

A further drawback to gastrectomy is that resections of this extent cannot be performed without some disturbance of digestion and disorder of the function of the upper alimentary tract, and these sometimes manifest themselves in the form of postgastrectomy syndromes such as dumping, regurgitation of bile, poor nutritional state, etc. (*see* page 191). These syndromes may occasion some discomfort in the early weeks after operation but generally improve markedly with the passage of time and in only a few cases do they persist in severe form. They are especially common and severe after the very *high* gastrectomies recently in vogue for duodenal ulcer; with lower gastrectomies they are perhaps less frequent, but the risk of recurrence of ulceration is of course greater.

To summarize then regarding the results of high gastric resection for duodenal ulcer,

juice to regurgitate through the stoma into the stomach and to neutralize to some extent the acidity of the gastric juice. By these two mechanisms most duodenal ulcers are caused to heal after posterior gastro-enterostomy. Unfortunately a certain percentage develop another ulcer in the jejunum adjacent to the stoma which causes more severe symptoms and more troublesome complications than the original duodenal lesion. Just how frequently stomal ulceration occurs after gastro-enterostomy is much debated. The estimates of different authorities have varied from 2–30 per cent or more (Walton, 1934, Wright, 1935, and Lowdon, 1948), and it is this variation that has resulted in gastro-enterostomy being both one of the most lauded and most condemned operations in surgery.

Undoubtedly some of the lower estimates have been due to an insufficiently long follow-up of patients, for stomal ulcer after gastro-enterostomy quite frequently does not develop for many years after operation; unless the cases are traced for at least 5–10 years many instances of recurrent ulcer will be overlooked. Another factor accounting for discrepancies in estimates of the incidence of stomal ulcer is the difference in the selection of cases for operation by different surgeons. The patients who are most likely to develop jejunal ulcers are the young or middle-aged individuals with high gastric acidity. After gastro-enterostomy their hyperchlorhydria often continues little abated and constitutes a serious threat to the relatively susceptible jejunal mucosa exposed at the stoma. If the operation is reserved for older patients with lower acidity, and particularly if their lesions have caused some duodenal stenosis, the incidence of jejunal ulceration is found to be much less though not completely eliminated. Comprehensive surveys of hospital statistics (as contrasted with the more partisan and less reliable reports of individual surgeons), such as those of Pyrah (1935) and of Cooper (1949), show that some 80 per cent of patients submitted to gastro-enterostomy for duodenal ulcer obtain good results. Of the other 20 per cent with further symptoms, 10 per cent come to a second operation for stomal ulcer, and 10 per cent, though having intermittent dyspepsia, are able to carry on—some of them with relatively little discomfort and reasonably satisfied with their results. The immediate mortality of gastro-enterostomy should be very low—not more than 1 per cent.

Dissatisfaction with the high incidence of jejunal ulcer after gastro-enterostomy has led most surgeons to abandon this operation as a routine procedure in the treatment of duodenal ulcer, though many have retained it as an occasional operation for poor risk elderly patients, especially those with stenosing ulcers. For the average case they have preferred to employ an extensive partial gastrectomy. However it has been argued by Walton (1930), who was one of the leading advocates of gastro-enterostomy, that it is more reasonable to employ this relatively safe operation as the routine procedure, and to reserve partial gastrectomy with its greater risks for the cases that develop stomal ulcer after gastro-enterostomy. Now that vagotomy has been introduced this argument is being advanced in support of the combination of gastro-enterostomy and vagotomy *vis-à-vis* gastrectomy.

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in a certain small proportion however—perhaps one in ten or fifteen—despite a careful dissection, the vagotomy is subsequently shown by the Hollander insulin test to be incomplete.

These three combinations of vagotomy and other procedures are all on trial at the moment. The operative mortality is essentially that of the associated procedure. *Vagotomy and pyloroplasty* thus carries a very small mortality. Pyloroplasty *per se* has been shown in the past to have little therapeutic effect on duodenal ulcer, so that cures after combined vagotomy and pyloroplasty must be attributed essentially to the vagotomy; this operation should therefore give a good indication of the curative value of simple vagotomy unhindered by retention phenomena. Recurrences have already been reported however by Tanner (1951) after this operation.

Vagotomy and posterior gastro-enterostomy, as already pointed out, is capable of healing most duodenal ulcers unaided, but it is liable to be followed by stomal ulceration in hyperchlorhydric patients, so that the real problem with combined vagotomy and gastro-enterostomy is whether the vagotomy will depress acidity sufficiently to protect the stoma from recurrent ulceration. If it could do this at any rate far enough to provide partial protection and reduce the incidence of stomal ulceration to a few cases in every hundred treated, this operation with its lower mortality might well replace subtotal gastrectomy as the standard operation for duodenal ulcer. Recurrences after it could be satisfactorily dealt with by a subsequent gastrectomy without subjecting all duodenal ulcer patients to the greater risks of this more major operation. However gastric analyses after combined vagotomy and gastro-enterostomy show that during the night or in response to a test meal or histamine injection there is still often sufficient acidity in the gastric contents for peptic digestion to take place, so that jejunal ulceration is theoretically possible (Farmer, Howe, Porell, and Smithwick, 1951). Long term clinical results are now becoming available for this operation and suggest that it may have little advantage over simple gastro-enterostomy. Thus Hoerr (1953), reporting from the Crile Clinic, Cleveland, on a series of 146 cases treated by vagotomy and gastro-enterostomy and followed up for from 3–5 years, records 85 per cent of satisfactory results, with 8 per cent of proven, and 4 per cent of suspected stomal ulcers, and an operative mortality of under 1 per cent. A collective survey by Jordon for the American Gastro-Enterlogical Association (1951) to determine the value of vagotomy and posterior gastro-enterostomy as contrasted with that of subtotal gastrectomy in the treatment of duodenal ulcer, gives some 75–80 per cent of satisfactory results with the former, but 87 per cent with the latter, the cases in both series having been followed for an average of two and a half years after operation. The operative mortality of gastro-enterostomy with vagotomy was only 1·4 per cent as compared with 2·5 per cent for gastrectomy.

Hemigastrectomy and vagotomy should abolish both neurogenic and gastrin-provoked secretions of HCl and result in a very severe depression of gastric acidity amounting almost to achlorhydria. Estimations of the acidity of the gastric contents adjacent to the stoma after this operation show that under all circumstances it is certainly less than that required for peptic digestion (*see* Farmer *et alia*, 1951). This operation ought therefore to offer a good insurance against recurrent ulceration, without depriving the patient of most of his stomach. On the other hand it would be surprising if the operative mortality of this procedure were much lower than that of an orthodox subtotal gastrectomy because the fatalities after gastrectomy are referable usually to accidents in

it should first be stressed that this operation has been used extensively in the last 15–20 years and that it is to be regarded as a well tried procedure, of which we know both the best and the worst. At the present day in good average hands a two-thirds gastrectomy for this condition is attended by an operative mortality of some three cases in every 100; of the survivors two or three will develop recurrent ulcers at the stoma, five will have *no further trouble from ulcer symptoms but will continue to have their results* marred to some extent by "dumping," by failure to put on weight and gain strength, etc , and ninety will, after some initial discomforts of the same kind, have excellent or very good results in all respects.

VAGOTOMY Though attempts have been made by Latarjet (1922) and others to treat peptic ulcer by division of the vagal nerve supply to the stomach, it was Lester Dragstedt (1945) of Chicago who introduced vagotomy as a rational measure in the *treatment of duodenal ulcer. He demonstrated that in patients suffering from duodenal* ulcer there is usually a gastric hypersecretion and hyperchlorhydria throughout the night. He postulated that, in the absence of chemical stimulation by food, this must be a neurogenic secretion presumably due to physical influences, and he was able to show that it could in fact be abolished by division of the vagi as they descend on the thoracic œsophagus. He claimed therefore that vagotomy corrected the essential abnormality of secretion in duodenal ulcer and was a logical measure in the treatment of this disease.

In practice however the operation was frequently found to be followed by undesirable complications, the most troublesome being *gastric retention* due to paralysis of the stomach from loss of its vagal supply, and *persistent diarrhoea.* As a result of the gastric retention (which was most severe in patients who had a certain amount of stenosis from the ulcer) vomiting and eructation of foul gas would occur, necessitating prolonged gastric aspiration via an indwelling Ryle's tube, and quite frequently a subsequent drainage operation, usually gastro-enterostomy, would be required. As for cure of the ulcer, the operation was successful in some 60 per cent of the cases (Johnson and Orr, 1953). Failures were sometimes referable to the fact that certain vagal fibres had escaped division, but occasionally they followed a vagotomy which was complete as shown by failure of the stomach to secrete HCl in response to hypoglycæmia down to 0·45 mgm. per cent induced by an injection of 20 units of insulin (the Hollander insulin test). An interesting complication of vagotomy for duodenal ulcer was the occasional development of a gastric ulcer some time later.

The frequent motor disturbances after *vagotomy alone* have led to the complete abandonment of this operation except in the treatment of a stomal ulcer following gastrectomy or gastro-enterostomy when the previous operation obviates the risk of retention. At the present time in the treatment of duodenal ulcer vagotomy is only used in conjunction with a drainage operation such as pyloroplasty, gastro-enterostomy or partial gastrectomy, of which gastrectomy is mechanically the most effective. The vagotomy is performed not through the chest but via the abdomen, the œsophagus being exposed by division of the peritoneum at the hiatus in the diaphragm and hooked down for two or three inches, rendering taut and easily identifiable by palpation the related vagal fibres which at this level are conveniently collected into two main trunks, an anterior and a posterior This is the "trans-hiatal supradiaphragmatic vagotomy." (For operative details *see* p 109) With experience of this technique it is possible to achieve complete division of the vagi in a considerable percentage of the cases;

Billroth I resection is virtually unknown, this operation is recommended as the procedure of choice in these cases.

Complications of Peptic Ulcer

Hæmorrhage. Minor degrees of bleeding are common in patients suffering from peptic ulcer, and occult blood can usually be demonstrated in the stools by chemical tests or spectroscopy. But in perhaps 12 or 15 per cent of the patients more severe hæmorrhage occurs resulting in macroscopic production of blood. If the ulcer is situated in the stomach the blood is usually vomited, sometimes in an unchanged condition but frequently altered by digestion so that the vomitus has a dark brown appearance like coffee grounds. Later, blood may also escape in the motions as melæna. If the hæmorrhage continues and is very severe, bright red blood will not only be brought up in the vomitus but may also appear in the motions. In duodenal ulcer, bleeding may not lead to vomiting but may be associated with a feeling of faintness which is later followed by the passage of a large black motion. Subsequently however the bleeding may be continued and become more severe, resulting in both hæmatemesis and further melæna.

In younger subjects even severe hæmorrhages are as a rule well borne but in elderly patients they may seriously endanger life. In these older subjects, who frequently suffer from coincident arteriosclerosis, major arteries eroded by deeply penetrating lesions are more likely to remain patent and continue bleeding; in addition these patients are less well able to stand a severe hæmorrhage. Thus Avery Jones (1947) found that the overall mortality in a series of 687 patients admitted to hospital with gastro-duodenal bleeding and treated medically was 7·8 per cent, but in patients over sixty years of age it was 15·8 per cent as contrasted with 3·3 per cent in those under that age.

The advisability of surgical treatment for bleeding peptic ulcer has been one of the most controversial issues in Medicine. Up to the end of World War II, despite eloquent pleas by Gordon-Taylor (1935) and Finsterer (1936) for operative intervention, the consensus of surgical opinion was opposed to operation for gastro-duodenal ulcer in the presence of severe hæmorrhage. Reliance was placed on the use of the Meulengracht regime of early feeding supplemented by blood transfusion, which often had to be continued for many days. Surgery was reserved as a last resort for very ill patients who had failed to respond to this conservative treatment, and the operative mortality under these conditions was naturally very high. In recent years, however, due largely to the work of Norman Tanner, there has been a considerable reorientation regarding surgical treatment of the bleeding ulcer. Tanner (1950) has shown that with good organization surgery need not be specially hazardous and for many cases the safest and most satisfactory treatment may be *early* operation. The cases for which surgery is specially indicated are obviously those with deeply penetrating chronic ulcers which have eroded major arteries producing a severe degree of hæmorrhage. It is not denied that even in these cases medical treatment may frequently be successful and that spontaneous arrest of bleeding may occur, but surgery probably offers a more certain prospect of saving life, particularly if the patient is elderly and suffers from arteriosclerosis.

However one of the great difficulties in the application of surgery is to select from amongst the cases admitted as emergencies with gastro-duodenal hæmorrhage those with the type of lesion just described, for only a proportion of these patients have in fact

connection with the duodenal stump and not to the height of the resection. However Johnson and Orr (1953) report a series of 236 cases of duodenal ulcer treated by vagotomy and moderate gastrectomy with an immediate mortality of 1·1 per cent and no recurrences to date.

It must be stressed that vagotomy in support of other gastric operations in the treatment of duodenal ulcer is still very much on trial. No operation for peptic ulcer can be properly evaluated till a substantial series of patients treated by it has been consistently followed up for at least five and preferably ten years. According to this criterion several more years will be required before the full value of these combined procedures can be determined, but, from the results so far recorded, it seems increasingly unlikely that they will displace subtotal gastric resection in the treatment of duodenal ulcer.

Operations for Gastric Ulcer

In the surgical treatment of gastric ulcer there is not the same overriding necessity to deal with gastric hyperacidity and hypersecretion that characterizes the surgery of duodenal ulcer. Indeed, the majority of cases with gastric ulcer have a normal or subnormal level of hydrochloric acid in the gastric juice. The most important principle in the surgical treatment of gastric ulcer is undoubtedly adequate excision of the lesion and the parts of the stomach in which ulcer is likely to form—namely the lesser curve and pyloric region. It is found that a moderate gastric resection achieves this and at the same time usually produces a profound depression of gastric acidity often amounting to complete anacidity, making recurrent ulceration most unlikely.

It should be noted that gastric ulcers have occasionally followed the performance of either gastro-enterostomy (Hurst and Stewart, 1929) or vagotomy (Johnson 1950), alone or in combination, for duodenal ulcer, indicating that these operations are unlikely to be curative for patients with established gastric ulcers. But there is a stronger argument than this against the use of these conservative measures in the treatment of gastric ulcer—it is that it is often difficult for the surgeon to be sure before operation or even at laparotomy whether an ulcer of the stomach is benign or malignant. It is therefore safer to practise excisional surgery for gastric ulcer, and thorough removal of the lesion whenever possible without undue risk may thus be taken as a first principle in the surgical treatment of this disease.

In the earlier days of gastric surgery ulcers of the stomach were frequently removed by various forms of strictly local excision—wedge excision, sleeve resection, or destruction by cautery—which were usually combined with gastro-enterostomy; but a straightforward gastrectomy has been found to give much better results and to be no more difficult technically than these lesser procedures. In the performance of a resection for gastric ulcer it is not necessary to sacrifice much of the greater curve or body of the stomach but the pyloric region and most of the lesser curve with fringes of adjacent anterior and posterior walls should be excised It is entirely optional whether the operation be completed by the Billroth II plan of anastomosing the gastric stump to the upper jejunum, or by the Billroth I technique of direct gastro-duodenal anastomosis, but gastric ulcers lend themselves particularly well to excision by the latter method, because a long duodenal stump extending right up to the pyloro-duodenal junction can always be prepared in these cases As disorders of alimentary function are possibly less common after the Billroth I type of resection and recurrence of gastric ulceration after adequate

Having regard to the foregoing considerations, the place of surgery in the emergency treatment of gastro-duodenal hæmorrhage may be defined thus:

(*a*) Patients with definite clinical or other evidence of a chronic ulcer who have a further severe hæmorrhage after admission or who continue bleeding over a period of 48 hours should be submitted to operation without further delay.

(*b*) Patients with no clear evidence of chronic ulcer will be treated conservatively in the first instance. If however they should have severe and alarming repetitions of hæmorrhage or continue bleeding less severely for five or six days the advisability of operation, in case a chronic ulcer is present, has to be seriously considered. The decision to employ surgery in these cases will be influenced by the age and general medical condition of the patient. In patients under the age of 50 it is usually safe to continue with medical treatment for a week or more unless the hæmorrhage is very severe, but in older patients, especially if arteriosclerotic, an earlier resort to exploratory laparotomy will be advisable.

It will be seen that the decision in favour of surgical treatment can be made soon in some cases but only after a period of observation in others. Clearly the closest possible co-operation between physician and surgeon is essential in the management of a patient with a gastroduodenal hæmorrhage and the surgeon should be called into consultation from the moment of admission, so that the subsequent course of the case may be a joint medico-chirurgical responsibility.

Operations for Bleeding Peptic Ulcer

These are among the most difficult in gastric surgery and should only be undertaken by an experienced surgeon, preferably one familiar with the technique of resection of duodenal ulcers, because this is frequently necessary. If really skilled surgical aid is not available it will often be preferable to continue with conservative treatment.

The patient comes to the theatre with a transfusion running, and there should be available ample supplies of blood, which may need to be given under pressure. The administration of a general anæsthetic to these patients carries a special danger because blood may be vomited during induction and may enter the larynx and bronchi. Even assiduous aspiration of the stomach through a Ryle's or large-bore stomach tube before and during the anæsthetic is no certain preventive of this disaster, because the blood in the stomach is usually partly clotted and blocks the eye of the tube. If a general anæsthetic is used therefore the anæsthetist should be alert to this hazard; he should induce anæsthesia with the utmost rapidity with thiopentone and a relaxant drug so that a tracheal tube with pneumatic cuff may quickly be inserted and the tracheo-bronchial tract protected from regurgitating stomach contents. Because of this danger of general anæsthesia some surgeons prefer to form these emergency operations under local abdominal analgesia and splanchnic block.

On opening the abdomen in the majority of cases an obvious chronic ulcer of the posterior duodenal or gastric wall or lesser curve will be discovered; *it is important* to bear in mind that more than one lesion may be present and that the bleeding may be coming from the less obvious of the two. But in other patients—often despite a clear history of a chronic peptic ulcer—no lesion of any kind can be detected in the stomach or duodenum. Examination of the liver and other organs may show a cirrhosis or other pathology, or may be entirely negative.

If an Ulcerative Lesion is Present a rapid partial gastrectomy is usually the best

a chronic ulcer. In the others the bleeding may be due to a variety of causes—superficial erosions of the gastric or duodenal mucosa, gastritis, gastro-staxis, hiatus hernia with congestion of the herniating part of the stomach, benign polyp or carcinoma of the stomach, œsophageal varices due to cirrhosis of the liver or other cause of portal hypertension, even to uræmia or hæmatological conditions. For some of these lesions such as gastric erosions or tumours an operation inadvertently undertaken may be usefully completed as a gastric resection, but generally the bleeding in these cases can be controlled by persistent medical treatment and operation merely subjects the patient to inconveniences and hazards of an unnecessary laparotomy. Surgery is therefore best avoided except when there is good reason for believing that a chronic ulcer is present. The main criteria for selecting cases of gastro-duodenal hæmorrhage for emergency surgery are as follows:

(1) Reasonable Evidence of the Existence of a Chronic Ulcer. The history in many cases will make it reasonably certain that they have a chronic gastric, duodenal or jejunal ulcer, and not a few are able to produce evidence from previous radiological examinations of the stomach and duodenum. Sometimes a barium meal examination carried out during the hæmorrhage may be successful in demonstrating an ulcer, but negative findings by this method are unreliable. Similarly gastroscopy in the presence of bleeding may disclose a gastric lesion, but again no significance can be attached to negative results. Finally it must be remembered that patients with chronic ulcers may occasionally give no history of ulcer dyspepsia so that if bleeding is very severe the surgeon may have to be prepared to accept the existence of a chronic lesion despite the absence of a typical history. It must also be mentioned that patients with a characteristic history of a peptic ulcer are occasionally found to have no chronic ulcer at laparotomy.

(2) Repetition or Continuation of the Bleeding. After admission to the ward the patient may improve remarkably with blood transfusion and morphia and there may be no further evidence of bleeding. On the other hand a fresh hæmatemesis may occur or there may be a sudden pallor associated with a rise in pulse rate and a fall in the blood pressure indicating a recurrence of bleeding into the intestine and followed later by the passage of melæna stools or even bright red blood per rectum. Further accelerations of the blood transfusion will be required to compensate for these losses and to restore the patient's circulatory condition, but in the course of two or three days there may be several alarming episodes of this kind, and in very severe cases the transfusion—even administered into two veins—may barely keep pace with the hæmorrhage. In yet other cases the bleeding may never be very severe but may continue as a slight loss over a period of several days or a week or two The recurrence of severe hæmorrhage or the continuation of bleeding over a period of a week or more are indications that a major artery has probably been eroded and may not seal itself off spontaneously.

(3) The Age and Coincidental Medical Condition of the Patient. As already mentioned, the age of the patient has a very important influence on the prognosis of a severe or continued gastro-duodenal hæmorrhage Older patients often have some degree of arteriosclerosis which renders them less well able to tolerate the cerebral anoxæmia of severe hæmorrhage, at the same time the rigid condition of the eroded artery makes it more difficult for it to contract What matters more than the patient's actual age is the condition of his cardiovascular system, but generally speaking the need to avoid continued or severe bleeding is greatest in patients over 50 years of age.

(*a*) Opening the stomach and duodenum by an anterior longitudinal gastroduodenotomy to look for a minute mucosal erosion or polyp which if present may be treated by gastrectomy or local excision. (*b*) Performance of a high partial gastrectomy, giving the patient a three-quarters or four-fifths chance of removal of any gastric erosion which may be responsible for the bleeding; and (*c*) Closure of the abdomen without further procedure to the stomach or duodenum, it being argued that any lesion too minute to be palpable will respond to medical treatment. The writer has known of patients with large impalpable superficial gastric erosions die under intensive medical treatment with massive transfusions, and he therefore prefers courses (*a*) or (*b*).

Perforation. This complication has become much more frequent in the last 15–20 years, being now at least four times as common as it was in the 1920s. It occurs most often with anterior wall duodenal ulcers. Posterior duodenal ulcers are more likely to penetrate into the pancreas or gastroduodenal artery and give rise to hæmorrhage; and ulcers of the lesser curve of the stomach are not so liable to perforate because they have to traverse the thicker gastric wall with its investing layer of lesser omental fat before reaching the peritoneum. Because of the much greater frequency of duodenal ulcer in males than females, most patients admitted with perforation are men. The perforation often occurs after a meal or a drink, though sometimes it takes place when the patient is fasting and even during sleep. Occasionally trauma in the form of an abdominal strain seems to play a part in the production of the perforation.

Usually the perforation is acute, coming on with dramatic suddenness and opening into the general peritoneal cavity. In other cases it is a more gradual process and a slight leak occurs leading to local peritonitis with the formation of adhesions, which may result in a spontaneous cure, or at most a localized abscess. Posterior wall perforations are liable to produce a localized peritonitis in the lesser sac. The ulcers which perforate are usually chronic, but this complication may also occur with acute ulcers. At the time of operation however the presence of surrounding œdema may make it impossible to be sure whether the original lesion was acute or chronic.

The effect of the escape of acid gastric contents into the peritoneal cavity is to produce a chemical irritation. This calls forth a secretion of fluid from the peritoneum which dilutes and neutralizes the acid. After 8–12 hours a bacterial peritonitis becomes established if the perforation is not closed.

Clinical Features of Perforation

The main symptom is the sudden onset of agonizing abdominal pain, usually more severe than anything the patient has ever experienced in his life before. The pain is situated initially in the epigastric region but later radiates over the entire abdomen. Occasionally the patient complains also of referred pain in one or both shoulder tips due to irritation of the peritoneum under the diaphragm. Vomiting may occur but it is not a constant feature. A history of previous indigestion of ulcer type is often forthcoming and not a few cases have perforated their ulcers before—sometimes two or three times. Some patients however resolutely deny any preceding gastric symptoms; but, when interrogated again during the calm of convalescence from operation, many of these cases with "silent ulcers" admit to occasional bouts of dyspepsia for some time before their perforation.

On examination the patient with a perforation usually lies in a rigid supine position

method of controlling the bleeding, and has the additional advantage of offering an almost certain cure for the patient's ulcer. In these cases gastrectomy will usually entail leaving the ulcer crater attached to the pancreas or liver and the main bleeding artery—the gastro-duodenal or splenic—will have to be underrun with a thread stitch as it appears in the crater. If the patient is bleeding severely it may be necessary for the surgeon to operate very rapidly till the ulcer crater has been exposed in this way and the bleeding point controlled. The gastrectomy is then completed more leisurely, leaving the crater outside the alimentary tract. The greatest difficulty may be encountered in dealing with deeply penetrating posterior duodenal ulcers, for, when these are bleeding, the methods of prepyloric section and resection for exclusion, either in its original form or in the Bancroft modification (*see* page 172), are quite inadmissible. Whatever may be the surgeon's usual procedure in disposing of an adherent posterior wall duodenal ulcer during gastrectomy he must in these cases resect to beyond the ulcer. A tedious dissection of the duodenal stump is thus usually necessary and the Nissen manœuvre for closure of the duodenal stump may be required (*see* page ~~172~~). 91

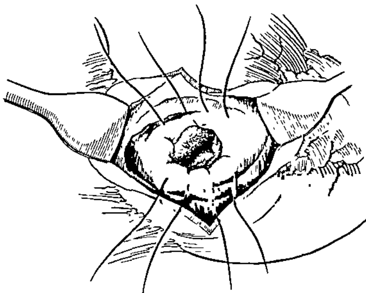


FIG 14. The method of dealing with a bleeding peptic ulcer by anterior gastrotomy, exposing the ulcer crater and undermining it with several deep catgut sutures These are tied tightly and the wound in the anterior wall of the stomach then closed

Occasionally, if the patient is very ill, as may happen when late surgical intervention is being undertaken, gastrectomy may be considered too hazardous and a simpler procedure providing direct control of the bleeding point may be preferred. This can be accomplished by incising the anterior wall of the stomach or duodenum, exposing the ulcer crater, and passing under it three or four deep non-absorbable sutures which when tied tightly effectively stop all bleeding (*see* Fig. 14). The opening in the anterior duodenal or gastric wall is then closed and in the case of a duodenal ulcer a posterior gastro-enterostomy can be added if the duodenum has been narrowed by this manœuvre. This operation is a good emergency procedure in desperately ill patients and is especially valuable for bleeding stomal ulcers where the alternative may be a particularly difficult gastrectomy. However occasionally the bleeding recurs again in some ten to fourteen days' time when the sutures have cut out Methods of trying to control ulcer bleeding by ligating main gastric vessels outside the stomach are quite futile, and the same is probably true of the operation of infolding and narrowing the duodenum by suture, combined with gastro-enterostomy for bleeding duodenal ulcer.

IF NO ULCER CAN BE DETECTED IN THE STOMACH OR DUODENUM (and no other pathology is found in the abdomen) there are three possible courses available to the surgeon:

(*a*) Opening the stomach and duodenum by an anterior longitudinal gastro-duodenotomy to look for a minute mucosal erosion or polyp which if present may be treated by gastrectomy or local excision. (*b*) Performance of a high partial gastrectomy, giving the patient a three-quarters or four-fifths chance of removal of any gastric erosion which may be responsible for the bleeding; and (*c*) Closure of the abdomen without further procedure to the stomach or duodenum, it being argued that any lesion too minute to be palpable will respond to medical treatment. The writer has known of patients with large impalpable superficial gastric erosions die under intensive medical treatment with massive transfusions, and he therefore prefers courses (*a*) or (*b*).

Perforation. This complication has become much more frequent in the last 15–20 years, being now at least four times as common as it was in the 1920s. It occurs most often with anterior wall duodenal ulcers. Posterior duodenal ulcers are more likely to penetrate into the pancreas or gastroduodenal artery and give rise to hæmorrhage; and ulcers of the lesser curve of the stomach are not so liable to perforate because they have to traverse the thicker gastric wall with its investing layer of lesser omental fat before reaching the peritoneum. Because of the much greater frequency of duodenal ulcer in males than females, most patients admitted with perforation are men. The perforation often occurs after a meal or a drink, though sometimes it takes place when the patient is fasting and even during sleep. Occasionally trauma in the form of an abdominal strain seems to play a part in the production of the perforation.

Usually the perforation is acute, coming on with dramatic suddenness and opening into the general peritoneal cavity. In other cases it is a more gradual process and a slight leak occurs leading to local peritonitis with the formation of adhesions, which may result in a spontaneous cure, or at most a localized abscess. Posterior wall perforations are liable to produce a localized peritonitis in the lesser sac. The ulcers which perforate are usually chronic, but this complication may also occur with acute ulcers. At the time of operation however the presence of surrounding œdema may make it impossible to be sure whether the original lesion was acute or chronic.

The effect of the escape of acid gastric contents into the peritoneal cavity is to produce a chemical irritation. This calls forth a secretion of fluid from the peritoneum which dilutes and neutralizes the acid. After 8–12 hours a bacterial peritonitis becomes established if the perforation is not closed.

Clinical Features of Perforation

The main symptom is the sudden onset of agonizing abdominal pain, usually more severe than anything the patient has ever experienced in his life before. The pain is situated initially in the epigastric region but later radiates over the entire abdomen. Occasionally the patient complains also of referred pain in one or both shoulder tips due to irritation of the peritoneum under the diaphragm. Vomiting may occur but it is not a constant feature. A history of previous indigestion of ulcer type is often forthcoming and not a few cases have perforated their ulcers before—sometimes two or three times. Some patients however resolutely deny any preceding gastric symptoms; but, when interrogated again during the calm of convalescence from operation, many of these cases with "silent ulcers" admit to occasional bouts of dyspepsia for some time before their perforation.

On examination the patient with a perforation usually lies in a rigid supine position

with the knees drawn up, the face pale and sweating, the expression anxious, the respirations thoracic in type with a short jerking grunting inspiratory phase, the alæ nasæ muscles active. The temperature is often subnormal but the pulse rate and blood pressure usually show no change at first. The abdomen is rigid and board-like and does not move with respiration; sometimes it is indrawn slightly above the umbilicus. On palpation it is diffusely tender but the tenderness is usually maximal in the epigastrium. The area of percussable liver dullness may be diminished, though this is a variable finding. Auscultation usually detects no peristaltic sounds in the patients with diffuse peritoneal irritation, but in cases of subacute perforation with localized peritonitis many loops of intestine may still be contracting normally, so that the abdomen may be far from silent on auscultation. A plain X-ray examination of the abdomen with the patient in a sitting position may show collections of gas as concentric shadows under the cupolæ of the diaphragm (*see* Fig. 15), but these may sometimes be absent despite a definite perforation.



(Reproduced by courtesy of Dr E Rohan Williams)

FIG 15. Radiograph, taken in sitting position, of patient with a perforated duodenal ulcer to show the gas shadows under both cupolæ of the diaphragm Note outline of spleen on L side

In the course of three or four hours, as the escaping gastric juice is diluted by the peritoneal exudate, the symptoms and signs often show a remarkable amelioration—*the stage of reaction.* The pain becomes less severe and the respirations easier; the pallor is replaced by a slight flush and the patient feels much improved. The abdominal tenderness and guarding may also diminish but as a rule they do not disappear entirely and are still important diagnostic signs. Also the pulse rate may now show some elevation.

This improvement however is short lived, and after a few hours the pain returns, the pulse becomes more rapid—up to 120 or more—and of poor quality, the temperature rises, and the abdomen becomes more tender and distended as peritonitis progresses.

With subacute perforation—the so-called "leaking ulcer"—the symptoms are less severe. The initial epigastric pain may not become generalized or may spread only to the right side of the abdomen due to the fact that the fluid escapes down the right paracolic gutter to the cæcal region. The symptoms and signs may disappear spontaneously in the course of several hours or a localized abscess may form. Many of these cases are dismissed in the first instance as exacerbations of ordinary ulcer pain, while a few with marked signs in the right iliac region may be mistaken for cases of acute appendicitis.

DIAGNOSIS

Usually the tender "boarded" abdomen makes the diagnosis obvious, but if the patient is first seen in the stage of reaction the true gravity of the condition may not be

appreciated. With an accurate history and a careful examination however it should be possible to exclude such conditions as biliary colic, acute appendicitis, acute pancreatitis, "diaphragmatic" pleurisy, or myocardial infarction. The features of special diagnostic importance are the suddenness of onset of the pain, the immobility of the patient and the abnormal tenderness and rigidity. Occasionally percussion of the area of liver dullness auscultation of the abdomen, or a radiological or electrocardiagraphic examination may be helpful in making a correct diagnosis.

Treatment

There is considerable controversy at the present time as to the correct treatment of perforated peptic ulcers. Three forms of management require to be considered, namely simple suture of the perforation, immediate partial gastrectomy, and a medical regime.

Simple Suture. Until comparatively recently this was the method of treatment universally adopted in this country. Its aim is to stop the continued infection of the peritoneal cavity by closure of the perforation and to save the patient from death due to peritonitis. It has the great advantage that it can be safely carried out by the relatively inexperienced surgeon, to whom this emergency often falls for treatment. The details of the operative technique are given on page 204. The operation itself carries little intrinsic risk, the outcome depending almost entirely on the length of time that has elapsed between the onset of the perforation and its suture. If the case is operated on within six hours there should be practically no mortality. The site of the perforation also influences the prognosis, for gastric perforations, especially those high on the lesser curve, are more often fatal than duodenal perforations. Older patients are naturally more liable to complications. These include all the complications that may follow any abdominal operation, but pulmonary collapse and broncho-pneumonia, empyema thoracis, and subphrenic or other form of residual abscess are especially common.

Simple suture is a purely life-saving procedure; it disregards completely the cure of the ulcer responsible for the perforation. Strangely enough however many patients enjoy a lengthy remission of their dyspeptic symptoms after suture of a perforation, and it was formerly believed that a considerable proportion of the cases were actually cured by this step. More prolonged follow-up studies by Illingworth, Scott, and Jamieson (1944) have established that the majority of the patients treated by simple suture of a perforated peptic ulcer suffer a return of their dyspepsia within five years, and that over 50 per cent of them come eventually to elective surgery.

Simple suture combined with posterior gastro-enterostomy. Often the duodenum is considerably narrowed when a perforation has been sutured and it might appear that trouble from stenosis subsequently would be inevitable. But with judicious use of gastric suction through a Ryle's tube post-operatively for a week or longer if necessary, till œdema in the region of the ulcer subsides, normal gastric emptying is usually resumed. Consequently a gastro-enterostomy to relieve anticipated stenosis is seldom indicated at the time of closure of the perforation and is better avoided because of the risk of subsequent stomal ulceration which seems to be specially great in perforation cases.

Primary Gastrectomy. On the Continent of Europe, following the lead of von Haberer (1919) and Yudin (1939), many clinics have adopted immediate partial gastrectomy as the treatment of choice for perforated peptic ulcers, simple suture being

reserved for the late cases in poor condition for which gastrectomy would be specially hazardous. The advantage claimed for this practice is that the patient is thereby not only cured of his perforation but also relieved of his disease. In the past it has seemed to British and American surgeons that this operation, performed as an emergency procedure, must subject the patient to quite unjustifiable risks. However, more recently several surgeons in this country have been exploring the possibilities of primary gastrectomy for perforated ulcers. They have been prompted to do so by the realization that the majority of the patients with perforated gastro-duodenal ulcers treated by simple suture come to elective surgery ultimately. Gastrectomy should not be practised in late cases in poor general condition, or in patients with perforation of acute ulcers or of chronic ulcers which did not cause severe dyspeptic symptoms. It is most strongly indicated in cases with a long history of chronic indigestion, especially if they have had a previous perforation. Most gastric ulcer perforations should probably be treated by immediate gastrectomy because, as Doll (1950) has shown, in 8 per cent of them the causal lesion is not a simple ulcer but a carcinoma, and the best chance of curing the patient probably lies with a primary resection. Most duodenal ulcers which perforate are situated anteriorly and are not adherent, so that gastrectomy in these cases is not as a rule technically difficult. None the less primary gastrectomy for a perforated ulcer should only be undertaken if the same surgical facilities and skill are available for its performance as for elective gastrectomy, and this proviso alone may considerably limit the use of this method of treatment.

If it should be shown eventually that vagotomy in combination with gastro-enterostomy or hemi-gastrectomy gives satisfactory late results for uncomplicated duodenal ulcer, the application of these alternative forms of elective surgery in the immediate treatment of duodenal perforations would have to be considered.

CONSERVATIVE TREATMENT has been recommended in recent years especially by Hermon Taylor (1951). It consists of passing a large bore stomach tube to evacuate the gastric contents by suction, and subsequently maintaining continuous aspiration of the stomach via a medium sized radio-opaque tube rather larger than a Ryle's tube. An initial X-ray of the abdomen is taken to determine the size of the subphrenic gas shadow and ensure that the tip of the tube is well down in the stomach. The patient is nursed in a semi-recumbent position and fluids and electrolytes are administered parenterally for two or three days. An hourly pulse chart is kept, the abdomen is frequently re-examined and radiological examination is repeated after 24 hours. If signs and symptoms increase or if there is any marked enlargement of the gas shadow on radiological examination surgery is advised. If, on the other hand, the clinical condition improves and the volume of gas diminishes, fluid feeds by mouth may be cautiously recommenced after 48 hours.

Undoubtedly many perforations can be successfully treated by this method, but it requires very close supervision and entails much more worry for the surgeon than does immediate suture. Its proponents only advocate it for early cases which usually make an uneventful recovery anyway after operative closure. The advantage claimed for conservative treatment is that the period of convalescence and absence from work is shorter after it than following operation but this seems a small consideration when weighed against the lessened anxiety and assured results after simple suture. In the writer's opinion conservative treatment should only be employed if circumstances are

such that emergency surgery cannot safely be practised, as, for example, in a small ship at sea, or in an isolated district abroad, remote from hospital facilities.

Fibrous Stenosis Following Ulcer. The fibrosis associated with a chronic or recurring ulcer may result in considerable contraction of the stomach or duodenum. In the case of an ulcer on the lesser curve this causes an hour-glass constriction of the stomach; with a duodenal or pyloric ulcer a simple duodenal or pyloric stenosis is produced.

Hour-glass Stomach

This is found with an ulcer of the middle third of the lesser curve which has caused abundant formation of scar tissue, extending into both anterior and posterior walls of the stomach. The greater curve is gradually drawn up towards the ulcer and eventually the fibrosis forms a tight constriction which divides the stomach into a proximal and a distal compartment. Owing to the obstruction to its emptying, the upper loculus is usually the larger, but in some cases a duodenal ulcer with stenosis is also present so that the lower loculus may also become distended.

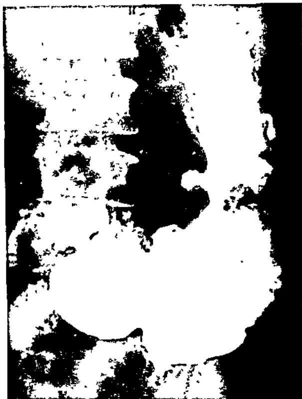


Fig 16 Radiograph after a Barium meal of a patient with large lesser curve ulcer producing an hour-glass constriction of the stomach

(Reproduced by courtesy of Dr E Rohan Williams)

Clinical Features. Patients with hour-glass constriction of the stomach are nearly always middle-aged or elderly females who give a long history of ulcer dyspepsia which has been accompanied by increasing vomiting, the vomitus usually being abundant and offensive. Pain may be present or absent, depending on whether the ulcer is active or healed.

On examination there may be signs of gastric dilatation, but the diagnosis of the condition depends on radiological examination after a barium meal. This outlines the two pouches, and the communication between them and may show an ulcer crater (*see* Fig. 16); sometimes a duodenal ulcer is also demonstrated.

Differential Diagnosis. The commonest cause of confusion is *spasmodic hour-glass contraction* where the constriction is due not to fibrosis but to spasm in association with a lesser curve ulcer. Radiologically the division of the stomach into pouches is less distinct and the spasm can often be overcome by an injection of atropin or banthine. *Malignant hour-glass contraction* due to gastric carcinoma usually causes a longer more irregular constriction between the pouches. *Post-operative adhesions* may occasionally constrict the stomach so as to cause an hour-glass deformity. *Diaphragmatic hernia* causes a constriction between the thoracic pouch and the remainder of the stomach but the radiograph shows that the stomach is displaced and the constriction coincides with the diaphragmatic shadow.

TREATMENT. Formerly operations such as gastro-gastrostomy to anastomose the two loculi and overcome the obstruction, or gastro-enterostomy to drain the upper pouch directly into the intestine were recommended for this condition. Occasionally in a particularly elderly emaciated patient with an hour-glass contraction one of these procedures, which can be rapidly executed with little shock, may still be advisable. For most cases however a partial gastrectomy is the procedure of choice. It has the advantage of removing the actual constriction which may contain an active ulcer.

PYLORIC OR DUODENAL STENOSIS

Stenosis from chronic pyloric or duodenal ulcer leads to dilatation of the stomach and hypertrophy of its wall. The patient gives a history of ulcer dyspepsia, with copious vomiting becoming the predominant symptom. Clinically there may be evidence of gastric dilatation in the form of splashing on succussion, or gastric peristalsis may be visible. The diagnosis is confirmed by radiological examination.

From frequent loss of fluid and chloride in the vomitus, dehydration, hypochloræmia, and alkalosis may result. The most severe form of alkalæmia in association with pyloric stenosis occurs when the patient in addition to vomiting, has been taking large doses of alkalis. Under these circumstances *gastric tetany* may be produced, causing carpopedal spasms and demonstrable hyperexcitability of the facial nerve. In cases of prolonged pyloric stenosis the fasting gastric juice may contain little free hydrochloric acid, but the total acidity is high due to the production of organic acids, such as lactic and butyric acid, by fermentation.

THE TREATMENT is surgical and follows the lines already discussed in the management of uncomplicated duodenal ulcer. Cases of this kind with a healed duodenal ulcer and marked cicatricial stenosis are recognized to be the best indication for simple gastro-enterostomy, but at operation it is generally impossible to be quite sure whether the ulcer is still active or not. Most surgeons therefore prefer to employ their usual operative treatment for duodenal ulcer—namely subtotal gastrectomy, or vagotomy plus either limited gastrectomy or gastro-enterostomy—and reserve gastro-enterostomy alone for specially elderly feeble subjects with stenosis.

In the preparation of these patients with pyloric stenosis—or hour-glass stomach—it is important to correct dehydration and electrolyte imbalance due to prolonged vomiting. In doing so it should be borne in mind that the depletion of chloride ions by loss of gastric HCl is not accompanied by a commensurate drain of sodium ions. The administration of saline which is frequently recommended in these cases to correct the chloride deficiency will therefore provide the patient with more sodium than he requires. Provided renal function is normal—but in these patients with stenosis who have had prolonged alkaline treatment it is often impaired—the excess of sodium ions may be satisfactorily excreted during the pre-operative period. If the infusion of saline is continued post-operatively however, the retention of sodium that ordinarily takes place during the first three or four days after major surgery may result in considerable imbalance. Some of the chloride for these cases with pyloric stenosis is thus better supplied in the form of a solution of ammonium chloride, the cation of which can be readily disposed of by the body without increasing the plasma sodium. The detailed management of the fluid and electrolyte disturbances in these cases requires close

biochemical supervision with frequent estimations of the concentration of the sodium potassium and chloride in the plasma.

In addition pre-operative gastric lavage should be carried out once or twice daily for several days before operation to overcome the local effects of the obstruction.

Jejunal or Stomal Ulcer

This form of ulceration occurs as an occasional complication after gastro-enterostomy or, more rarely, after gastrectomy, but only when these operations have been performed for duodenal ulcer, never after short-circuit or resection for gastric carcinoma, and practically never after these procedures for gastric ulcer. As already indicated, it has played an important role in determining the choice of operative procedure for duodenal ulcer.

Jejunal ulcers are usually situated in the first two inches or so of the efferent limb of the anastomosed jejunal loop or in the jejunum opposite the stoma, and in the great majority of cases the ulcer actually extends proximally as far as the suture line between the stomach and jejunum. Rarely does the ulcer lie on the gastric side of the anastomotic line. The lesion has the appearance of any other peptic ulcer and exhibits the same tendency to produce hæmorrhage, perforation or fibrosis. Perforation may occur into the general peritoneal cavity, or, quite frequently, is of a more chronic nature, giving rise to a localized abscess in the vicinity of the anastomosis or in the lesser sac, or penetrating into the transverse mesocolon and colon and producing a gastro-jejuno-colic fistula.

Originally it was thought that jejunal ulcers were due to technical faults at the original operation—such as damage to the jejunum by occlusion clamps, excision of gastric mucosa, or the use of a non-absorbable suture material—but there is no clear evidence that these play any significant rôle. The one fairly constant feature about these cases of jejunal ulcer is that they have had a severe hyperchlorhydria before operation and that this usually persists afterwards, and as this state of affairs is most commonly found in young or middle-aged male patients jejunal ulcer occurs more frequently in these age groups than in older patients; it is rare in women at any age. It is said to be especially common after operations in which an entero-anastomosis is made between the limb of the jejunal loop, or the anastomosis between stomach and jejunum is made on the Y principle advocated by Roux (1897). Either of these manœuvres has the effect of draining bile and pancreatic secretion away from the part of the jejunum sutured to the stomach, thus allowing unneutralized gastric juice to come in contact with jejunal mucosa.

Clinical Features

The development of a jejunal ulcer usually proclaims itself by causing a recurrence of the patient's pre-operative pain, and the occurrence of genuine pain—as contrasted with a feeling of epigastric fullness—after an operation for peptic ulcer is always presumptive evidence of further ulceration. Sometimes the pain has much the same characteristics as a duodenal ulcer pain, but it is often situated more to the left and lower down in the abdomen opposite the umbilicus; it is also frequently more severe and continuous. Vomiting is not uncommon and bleeding in the form of melæna or hæmatemesis is an important symptom. The symptoms may come on soon after operation and many patients who are subsequently shown to have jejunal ulcers admit on

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again after a truly subtotal resection, total gastrectomy had to be considered—a serious step indeed. But now vagotomy is available for these cases. It can be performed through the abdomen, or, if adhesions from previous operations make this difficult, a transthoracic vagotomy may be employed instead. No large series of cases so treated and followed for a long time has yet been published, but for stomal ulcer following an adequate partial gastrectomy simple vagotomy is undoubtedly the correct treatment. Where a very limited resection has been performed and a large gastric stump remains it is probably wise to carry out a proper subtotal gastrectomy as well as performing vagotomy. For recurrence after an adequate partial gastrectomy and vagotomy the only effective operation available is a total gastrectomy.

Gastro-jejuno-colic Fistula. About 10 per cent of all jejunal ulcers that present for surgical treatment are complicated by the development of a fistula into the colon. This results in severe diarrhœa, fæcal vomiting and a rapid decline in the patient's general condition due to dehydration and electrolyte depletion and to lack of nourishment. It is interesting that the pain of the jejunal ulcer usually disappears completely when a fistula develops. Untreated the condition is inevitably fatal in a few weeks or months. The diagnosis can usually be made by the characteristic clinical features and is confirmed by radiological examination after a barium meal and enema.

Surgical Treatment

This is urgently required as soon as the patient can be brought into fluid and electrolyte balance by intravenous infusions and anæmia can be improved by blood transfusion. Formerly it was the practice to separate the colon from the stomach and small gut, close the opening in the former and carry out an extensive gastrectomy with resection of the loop of jejunum containing the ulcer. This was technically difficult and time-consuming and to these debilitated patients represented a formidable procedure carrying a high operative mortality. Pfeiffer (1939) has shown that the diarrhœa in this condition is due not to the passage of food from the stomach into the colon as was formerly thought, but rather to the entry of colon contents into the small gut through the fistula with the production of a severe enteritis. It can be completely eliminated by the establishment of a loop colostomy proximal to the fistula, that is in the right end of the transverse colon or in the upper part of the ascending colon. After this step the patient's general health improves enormously and some weeks later the surgeon may make a direct attack on the gastro-colic fistula with much less risk. At this second operation the colon is separated from the stoma, partial colectomy being performed if necessary, and a high partial gastrectomy is carried out with resection of the piece of jejunum containing the ulcer. A vagotomy may be added if desired.

TUMOURS OF THE STOMACH

Benign Tumours

According to Eusterman and Senty (1922) less than 1 per cent of all gastric neoplasms are benign, but careful pathological examination may reveal tiny polyps more frequently. Benign tumours may arise from any of the layers of the gastric wall and may form either flat plâque-like tumours or, more commonly, large protuberant growths which project into the lumen of the stomach or on its external surface. The following types are described:

retrospective questioning that they had occasional slight dyspepsia long before they reported to hospital again. But other patients, especially after gastro-jejunostomy, may remain symptom free for 5 or 10 years before symptoms of a jejunal ulcer appear. In a large series of cases of jejunal ulceration Gibson and Priestley (1948) found that the average interval that elapsed between operation and the diagnosis of a jejunal ulcer was 3½ years after gastro-enterostomy, but only 2¼ years after partial gastrectomy. *Occasionally the first real manifestation of the condition may be the occurrence of a hæmorrhage or a perforation.*

Clinical examination is usually negative though sometimes there may be a palpable inflammatory mass in the vicinity of the anastomosis. The demonstration of a persisting hyperchlorhydria on gastric analysis is suggestive and the detection of occult blood in the stools is valuable supporting evidence. The radiologist can sometimes detect an ulcer niche or elicit tenderness in the region of the stoma and note delay in emptying through it. A jejunal ulcer can sometimes be seen by the gastroscope when radiology has been negative, but the technical difficulties of gastroscopy in the post-operative stomach are often such as to make a satisfactory viewing of the stomal region impossible. In many cases of jejunal ulcer radiological and gastroscopic examination is negative and the condition must be diagnosed essentially on the history of recurrent ulcer pain.

Treatment

This may be medical if the symptoms are slight, and under these circumstances indeed the patient will probably be reluctant to contemplate further surgery. In most cases however medical treatment is unavailing and operation is required, and in the face of continued symptoms it should not be long delayed because the risks of complications with a jejunal ulcer are considerable.

Operations for Jejunal Ulcer Arising After Gastro-Enterostomy

The treatment adopted by most surgeons for an established jejunal ulcer after gastro-enterostomy is a high partial gastrectomy with resection of the piece of jejunum attached to the stomach. This is generally a more difficult operation than a gastrectomy for a duodenal or gastric ulcer, and in the past has been regarded as especially dangerous. As the report of Gibson and Priestley (1948) from the Mayo Clinic shows, this is no longer true; in their series of cases there was an operative mortality of 2·9 per cent, and 87·5 per cent of the survivors had satisfactory results 5—10 years after the operation.

Since the introduction of vagotomy, many cases of stomal ulcer after gastro-enterostomy have been treated by this operation Good results have been claimed, but from the inquiry of Jordon (1951) for the American Gastro-Enterological Society on the value of vagotomy in the treatment of jejunal ulcer, it is doubtful whether these are as good as the results yielded by subtotal gastrectomy. Probably the majority of surgeons still employ gastric resection in the treatment of these cases, but many of them now combine vagotomy with it in the hope that this will give the maximum protection against recurrence.

Operations for Jejunal Ulcer Arising After Partial Gastrectomy

Before 1944 the surgical treatment of these cases consisted of a further partial gastrectomy if that were possible, but if little stomach remained or if the ulcer recurred

and the correct procedure. Multiple benign polypi in the pyloric region are best treated by partial gastrectomy, as there is strong evidence that this is a precancerous condition.

SARCOMA OF THE STOMACH

Sarcomata are comparatively rare tumours of the stomach and probably represent not more than 1 per cent of all gastric neoplasms (Walton, 1930). They may occur at almost any age, including childhood, but are commonest between thirty and fifty. The sexes are affected equally. The tumour originates in the submucosa or muscularis often towards the pyloric region, and either infiltrates the wall of the stomach, causing great thickening, or forms a polypoid swelling projecting into the gastric lumen or the peritoneal cavity. Central necrosis of the growth may lead to ulceration and hæmorrhage or occasionally to cyst formation. Histologically three varieties of gastric sarcomata can be recognized: the round celled sarcoma (which may be confused with an anaplastic carcinoma), the spindle celled sarcoma, and, by far the commonest, the lymphosarcoma. Metastases may occur in the liver or the related lymphatic glands, the latter being especially frequent with lymphosarcomata.

Clinical Features

The symptoms do not differ essentially from those of a gastric carcinoma but vomiting is not so frequent as in the latter condition and a palpable mass is even more common. Achlorhydria is usual though not invariable. Radiologically the projecting endogastric sarcoma shows as a smooth rounded filling defect which may be mistaken for a polypoid carcinoma or a benign tumour, whilst the infiltrating variety of sarcoma may mimic a diffusely invasive carcinoma. Gastroscopy is apt to be equivocal in these cases. Some gastric sarcomata which project into the peritoneal cavity may cause no dyspeptic symptoms at all, but may present as a puzzling abdominal swelling.

Treatment

Sarcomata of the stomach are often amenable to cure by gastrectomy, and unless the growth has obviously spread beyond the scope of surgical eradication, or the general condition contra-indicates operation, exploratory laparotomy should be performed. It may then be found that hepatic or lymphatic metastases render excision impossible but two thirds of Balfour and McCann's (1934) cases were suitable for gastrectomy. Roughly a third of the operation survivors lived for five years. X-ray treatment is a useful post-operative adjunct after removal of lympho-sarcomata, because they are very sensitive to radiotherapy.

CARCINOMA OF THE STOMACH

Incidence

Carcinoma of the stomach is a disease of appalling frequency and deadliness. The Registrar-General's reports show that it is still the commonest form of malignant disease encountered in this country—though carcinoma of the bronchus bids fair to outstrip it in the near future. Every year in England and Wales some 15,000 persons lose their life from gastric cancer. Moreover despite the gradual shrinkage of our population there has been a steady increase in the number of deaths from cancer of the stomach, as of most other organs, during the past 25–30 years, probably due in large

Myomata and Fibromyomata. These are said to be the commonest benign tumours. They consist of unstriped muscle mixed with a varying amount of fibrous tissue, are normally single and generally originate near the curvatures of the stomach or towards the pylorus. They may reach a large size.

Adenomata and Papillomata. These are usually small soft rounded pedunculated or sessile tumours arising most frequently in the pyloric region and projecting into the lumen. They may be single or, more commonly, multiple. Sometimes they occur in association with carcinoma of the stomach. Stewart (1931) moreover has found that 28 per cent of all polyps show carcinomatous change. It seems likely therefore that adenoma may sometimes be an intermediate stage in the development of gastric cancer.

When the polypi are especially numerous the condition is termed *gastric polyposis*. Sometimes the polypi in this condition are confluent and form a well defined thickened plâque in the gastric wall.

Neurofibromata and Neurolemmomata. *Neurofibromata* are always part of a generalized von Recklinghausen's disease with cutaneous fibromata, pigmented spots, skeletal deformities, etc. The lesions in the stomach are multiple small tumours usually projecting into the lumen.

Neurolemmoma on the other hand is usually unassociated with other nerve lesions. It occurs singly and forms a large polypoid mass projecting into the stomach cavity. Often there is a central mucosal defect on the summit of the swelling; this may deepen and hæmorrhage may take place from it.

Hæmangioma. This is a rare tumour in the stomach and gives rise to a localized bluish-black discoloration of the gastric wall which is liable to bleed.

Lipomata. These may form as small localized collections of fat in the subserous or submucous layer Rarely do they enlarge to produce big projecting tumours.

Clinical Features. These tumours may be entirely "silent" or may cause vague dyspepsia. Not infrequently they produce hæmorrhage which may be severe and may be the mode of presentation. Very rarely the tumour may initiate an intussusception through the pylorus or a volvulus of the stomach. The tumour may be palpable clinically if of large size. Radiological examination of the larger tumours shows usually a sharply defined filling defect with a smooth surface, often with a punched out niche on its summit, no evidence of infiltration of the gastric wall is seen and the mucosal folds appear normal on screening. Gastric polyposis on the other hand causes numerous smooth closely grouped filling defects arranged like clusters of grapes, usually in the pyloric region Gastroscopy will confirm that the tumour is covered with normal mucosa and therefore presumably benign, but it does not offer any other assistance in deciding on the nature of the growth, except that hæmangiomata can usually be recognized by their colour. Usually the precise diagnosis depends on exploratory operation, and the patient comes to laparotomy on a tentative diagnosis of carcinoma of the stomach.

Treatment. If from the softness of the tumour at operation and the fact that it is seen to be covered with intact mucous membrane on opening the stomach, the surgeon feels confident that it is benign, a localized resection of the affected part of the stomach is justifiable; but often it is impossible to be certain. Under these circumstances a partial gastrectomy should be carried out as for carcinoma. Sometimes frozen sections may help the surgeon to make up his mind as to the nature of the growth at operation

and the correct procedure. Multiple benign polypi in the pyloric region are best treated by partial gastrectomy, as there is strong evidence that this is a precancerous condition.

SARCOMA OF THE STOMACH

Sarcomata are comparatively rare tumours of the stomach and probably represent not more than 1 per cent of all gastric neoplasms (Walton, 1930). They may occur at almost any age, including childhood, but are commonest between thirty and fifty. The sexes are affected equally. The tumour originates in the submucosa or muscularis often towards the pyloric region, and either infiltrates the wall of the stomach, causing great thickening, or forms a polypoid swelling projecting into the gastric lumen or the peritoneal cavity. Central necrosis of the growth may lead to ulceration and hæmorrhage or occasionally to cyst formation. Histologically three varieties of gastric sarcomata can be recognized: the round celled sarcoma (which may be confused with an anaplastic carcinoma), the spindle celled sarcoma, and, by far the commonest, the lymphosarcoma. Metastases may occur in the liver or the related lymphatic glands, the latter being especially frequent with lymphosarcomata.

Clinical Features

The symptoms do not differ essentially from those of a gastric carcinoma but vomiting is not so frequent as in the latter condition and a palpable mass is even more common. Achlorhydria is usual though not invariable. Radiologically the projecting endogastric sarcoma shows as a smooth rounded filling defect which may be mistaken for a polypoid carcinoma or a benign tumour, whilst the infiltrating variety of sarcoma may mimic a diffusely invasive carcinoma. Gastroscopy is apt to be equivocal in these cases. Some gastric sarcomata which project into the peritoneal cavity may cause no dyspeptic symptoms at all, but may present as a puzzling abdominal swelling.

Treatment

Sarcomata of the stomach are often amenable to cure by gastrectomy, and unless the growth has obviously spread beyond the scope of surgical eradication, or the general condition contra-indicates operation, exploratory laparotomy should be performed. It may then be found that hepatic or lymphatic metastases render excision impossible but two thirds of Balfour and McCann's (1934) cases were suitable for gastrectomy. Roughly a third of the operation survivors lived for five years. X-ray treatment is a useful post-operative adjunct after removal of lympho-sarcomata, because they are very sensitive to radiotherapy.

CARCINOMA OF THE STOMACH

Incidence

Carcinoma of the stomach is a disease of appalling frequency and deadliness. The Registrar-General's reports show that it is still the commonest form of malignant disease encountered in this country—though carcinoma of the bronchus bids fair to outstrip it in the near future. Every year in England and Wales some 15,000 persons lose their life from gastric cancer. Moreover despite the gradual shrinkage of our population there has been a steady increase in the number of deaths from cancer of the stomach, as of most other organs, during the past 25–30 years, probably due in large

measure to the fact that people are living longer and being exposed to the risks of developing cancer that attend advanced age. There is certainly no ı anticipate any change in the trend of incidence in the immediate future.

According to Harnett (1947) carcinoma of the stomach occurs most fr between the ages of 45 and 75 years and is twice as common in men as in women is a marked racial and geographical variation in the incidence of the disease. is commonest in white peoples and very rare in most coloured races. The incid England is only about half that in most Western European countries, and there to be a lower incidence in Southern England than in Northern England or Scc Though a striking familial tendency to gastric cancer is sometimes apparent, it h been possible to establish this on a valid statistical basis.

Ætiology

Little more is known of the cause of cancer of the stomach than of malignant dis in general, but certain factors do seem to be of ætiological significance:

Experimental Data. The Danish worker Fibiger (1913) claimed that by feeding on cockroaches infected with the gastric nematode *Gangylonema neoplasticum*, chro irritation of the stomach could be produced resulting in the development of chro gastritis, polyposis, and eventually carcinoma. Subsequent investigators such as Pass Lees, and Knox (1936) however incline rather to the view that Fibiger's results were d to a hyperkeratosis resulting from a vitamin-A deficiency in the diet.

There is experimental evidence for the view that ingestion of superheated fat ma provoke cancer of the stomach in the mouse or rat (Peacock, 1947). It would appea that heating the fat to a temperature above 250 degrees results in the production of carcinogenic substances. Administration of one of these substances, such as methylcholanthrene, either mixed with the food or by injection into the gastric mucosa, has caused squamous celled carcinoma in the stomach or forestomach of susceptible mice. Strong (1945), working with hybridized mice, has succeeded in perpetuating the carcinogenic effect of methylcholanthrene through subsequent generations of mice without further administration of the carcinogen. Excellent reviews of the experimental work on the production of gastric cancer have been published by Klein and Palmer (1941) and Barrett (1946).

Clinical Precursors:

(1) Adenomatous Polyps

It would seem that, as with the colon and rectum, adenomatous polyps may occasionally be the starting points for malignant disease of the stomach, but the explanation of the hyperplasia leading to the former and culminating in the latter has still to be found.

(2) Chronic Gastritis and Pernicious Anæmia

It has long been debated whether chronic gastritis in the human may predispose to the development of gastric cancer, in the same way as chronic colitis has now been established to do for colonic cancer (*see* page 67). Unfortunately gastritis is a much less definite clinical entity than ulcerative colitis and does not call for surgical treatment, so that it is difficult to secure accurate clinical or pathological data on this possible malignant predisposition.

One form of gastritis however does present in an easily recognized form and is amenable to careful follow-up study, and that is the atrophic gastritis found in association with pernicious anæmia. Kaplan and Rigler (1945) subjected 293 patients with achlorhydria and gastritis in combination with pernicious anæmia to repeated clinical and radiological examinations, and reported that 36 or 12 per cent developed carcinoma of the stomach. This incidence would seem to be higher than the incidence that might be expected in the ordinary population of the same age and sex distribution; and presumably further follow-up of these cases will reveal additional carcinomata. Wilkinson (1950) in this country, however, has also reviewed a large series of patients with pernicious anæmia and is not convinced that they show any unusual predisposition to the development of gastric cancer.

(3) Chronic Gastric Ulcer

Another chronic irritative process found in the stomach is a chronic peptic ulcer, and the possible predisposition to malignant degeneration engendered by such a lesion requires to be examined. In some ulcerating carcinomata of the stomach the gross appearance may closely resemble that of a benign peptic ulcer, and the histological examination in these and other more typical gastric cancers may show evidence of pre-existing simple chronic ulceration. It has been claimed by Wilson and McCarty (1909) of the Mayo Clinic that at least 70 per cent of all gastric carcinomata show such evidence, but most pathologists in this country have been unable to confirm this very high estimate. Thus Dible (1924) in a very careful investigation of cases of gastric cancer found no indication whatsoever of pre-existing peptic ulceration in 84 per cent, and in the remaining 16 per cent considered the evidence on this point completely equivocal. Newcomb (1933) and Stewart (1931) reported that 12–16 per cent of the specimens of malignant disease of the stomach examined by them showed histological signs of previous chronic peptic ulceration and were to be regarded as examples of *carcinoma-ex-ulcere* or *ulcer-cancer*.

Bearing on this problem is the related one as to how frequently an apparently benign gastric ulcer undergoes malignant change. Many simple gastric ulcers on section show near their margins groups of epithelial cells arranged in small clusters or irregular tubules. Some pathologists have regarded these cells as evidence of malignant change and as a consequence have formed the opinion that ulcers frequently become carcinomatous. Others consider these to be normal epithelial cells displaced by fibrosis and contraction of the gastric wall. Moreover, a high estimate of the frequency of carcinomatous change in simple peptic ulcer of the stomach accords ill with impressions derived from the conservative clinical management of patients suffering from this latter condition. The most striking clinical evidence on this point is that reported by Balfour from the Mayo Clinic. It used to be the practice in that institution to treat apparently benign gastric ulcers by simple gastro-enterostomy, the ulcer itself being untouched surgically. On a long-term follow-up extending over many years, of 1,200 cases treated in this way Balfour found that only 6 per cent had developed carcinoma of the stomach, and it is highly likely that in most of these patients the lesion was malignant *ab initio* and wrongly diagnosed at operation. It may be said therefore that whilst chronic gastric ulcer undoubtedly may undergo carcinomatous change, the incidence of this development is probably very low, certainly less than 6 per cent.

Pathology

Primary gastric carcinoma may occur in any part of the stomach but originates more frequently in the pyloric region than elsewhere. According to Willis (1953) the relative incidence of growths in the different parts of the stomach as determined in a large series of necropsies is as follows.

Pyloric region . . .	474 = 47 per cent
Lesser curve . . .	262 = 26 ,, ,,
Cardia . . .	103 = 10 ,, ,,
Rest of stomach . .	89 = 9 ,, ,,
Whole stomach . . .	78 = 8 ,, ,,
	1006

Though double or multiple primary carcinomata have been described in the stomach the growth is usually single and presents in one of the following forms (It must be understood however that these are not hard and fast distinctions and that some growths exhibit the features of more than one type):

(1) **The Polypoid or Fungating Growth.** This forms a big soft cauliflower-like mass protruding into the lumen of the stomach (*see* Fig. 17). If situated in the body of the stomach it may reach a large size without interfering with the passage of the gastric contents, but if, as is more common, it occurs in the pyloric region it may soon cause obstruction. The surface of the tumour rapidly undergoes necrosis leading to ulceration, hæmorrhage, and cachexia. Spread through the wall of the stomach is a slower process as a rule than with deeply ulcerating growths so that results of surgical excision are generally better than with other forms of gastric cancer.



FIG 17 Specimen of stomach showing polypoid carcinoma growing from the posterior wall of the pyloric region and projecting into the lumen

Histologically these growths are usually found to be composed of columnar cells arranged in tubules but sometimes a spheroidal celled form is encountered.

(2) **The Colloid or Mucoid Growth.** This is a relatively rare type of gastric carcinoma in which colloid degeneration has taken place with the production of large quantities of mucus-like material in the cells, acini and tissue spaces. The malignant cells become bloated and undergo a displacement of their nuclei to one side so that they present a "signet-ring" appearance. The growth is usually found in the pyloric region, it tends to infiltrate the stomach wall widely, causing much thickening and giving it a peculiar translucent appearance. It seldom invades the mucous lining of the stomach but soon reaches the peritoneal surface and gives rise to widespread peritoneal metastases and involvment of adjacent organs, which may be gummed together by thick gelatinous material.

(3) **The Diffuse Leather-bottle Stomach; Linitis Plastica or Fibromatosis of the Stomach.** In this form the gastric wall, first usually in the pyloric region, later in the body and finally sometimes throughout the entire stomach, undergoes a diffuse thickening and induration often measuring 2 cm. across (*see* Fig. 18). This is due mainly to fibrosis in the submucous and subserous layers; the muscle coat presents a remarkable appearance on section, for the circular fibres are seen to be greatly hypertrophied and separated by fibrous bands, the appearance being termed segmentation of the muscle. The serosa overlying the thickening is usually pearly white in colour. The mucosa is usually thrown into deep folds due to the contracted condition of the organ. It is œdematous and shows catarrhal changes, and in many cases it contains an ulcer in the pyloric region, which is presumed to be the starting point of the growth. As a result of the changes in its wall the stomach becomes greatly contracted and its capacity may be reduced to a few ounces. Usually there is no stasis and food passes through it with great rapidity.

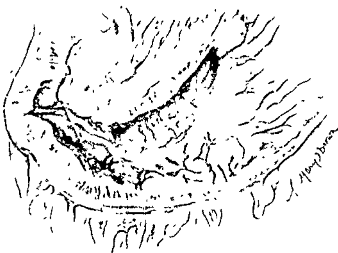


Fig 18 Specimen of stomach the seat of a linitis plastica—"leather-bottle" stomach

Microscopic examination discloses a dense fibrosis of the gastric wall in which are buried groups of spheroidal cancer cells. Sometimes many sections have to be examined before any cells are discovered. Failure to detect malignant cells at all in such cases had led to the idea that a simple fibromatosis of the stomach may exist, but it is probable that all these cases are malignant.

Lymphatic metastases may occur late in the course of the disease. Hepatic deposits are likewise often long delayed.



Fig 19 Specimen of ulcerating carcinoma of the pyloric part and body of the stomach

(4) **The Ulcerating Carcinoma.** This is the commonest type of gastric cancer and may occur anywhere in the stomach but is most frequently found in the pyloric region or on the lesser curve. Usually it presents the appearances of a typical malignant ulcer,

with a shallow irregular crater lined by necrotic malignant tissue, the edges being hard, raised and nodular. The base of the ulcer and the surrounding gastric wall may be much indurated. The serosa may be relatively normal in the early stages but later is usually invaded by the growth and is hard, white and nodular. When the part of the stomach containing the ulcer is cut across it is seen that the entire ulcer is raised above the surrounding mucosa and really projects into the lumen of the stomach, this being in sharp contrast with what obtains in simple peptic ulcer (*see* Fig. 19). Sometimes however an ulcerating carcinoma may closely resemble a benign peptic ulcer except that at one point on the circumference of the lesion more typical appearances of a new growth are seen in the form of a raised nodular edge or infiltration of the gastric wall.

Microscopically these growths are usually seen to be composed of spheroidal cells arranged in clusters in a fibrous stoma, but sometimes an alveolar formation is found.

Secondary Gastric Carcinoma. This results from spread of carcinomata of adjacent viscera such as the œsophagus, gall-bladder, pancreas, or transverse colon. Many growths of the upper part of the stomach involving the cardia turn out to be squamous epitheliomata derived from the œsophagus.

Spread of Carcinoma of the Stomach

Direct Spread. Any form of carcinoma of the stomach as it increases in size, spreads by direct continuity through the substance of the gastric wall. Infiltration of the submucosa may be apparent to the naked eye as white streaks of growth at the periphery of the primary lesion, and around this again is a zone of microscopic spread in the intramural, chiefly submucous, plexus of lymphatics of the stomach wall. This extension beyond the palpable and evident edge of the primary lesion is often 1–1½in. wide according to Coller, Kay and McIntyre (1941).

The gross spread of gastric cancer takes place in all directions but apparently stops short at the pyloroduodenal junction. On microscopical examination, however, as Castleman (1936) and Zinninger and Collins (1949) have shown, the lymphatics of the duodenal wall may be seen often to contain carcinoma cells to a distance of 1 in. or more beyond the pylorus, so that resected specimens of gastric carcinoma not infrequently contain microscopic evidence of growth at the distal line of resection.

At the proximal end of the stomach there appears to be no hindrance whatsoever to the invasion of the œsophageal wall by direct extension of gastric carcinomata—or of the gastric wall by œsophageal growths. Both macroscopic and microscopic spread occurs with the greatest of ease, the latter sometimes to a distance of 4 or 5 cm. according to Eker (1951).

Direct spread of carcinoma of the stomach also leads to *penetration of the gastric wall* so that the peritoneal surface is eventually invaded. Further extension may then take place in two ways:

(*a*) By Transcœlomic Spread Through the Peritoneal Cavity

Carcinoma cells break loose from the peritoneal aspect of the growth and mostly descend under the influence of gravity towards the pelvis. They may become engrafted on other peritoneal surfaces giving rise to nodules or plâques of growth on the parietal peritoneum, greater omentum, and other viscera, notably the pelvic organs. Sometimes large tumours are produced in this way in the ovaries—the Krukenberg tumours—and

sometimes when the primary gastric lesion is small the pelvic condition may be regarded as a primary ovarian tumour.

(*b*) BY SPREAD TO ADJACENT ORGANS

Quite frequently the peritoneal surface of the stomach overlying the growth becomes adherent to neighbouring organs such as the pancreas, liver, omentum, transverse colon, or mesocolon, or small gut, before the malignant process actually reaches it. In due course the growth will invade these organs and be disseminated by their lymphatic system in addition to that of the stomach itself, but in the earliest stages the adhesions are often purely inflammatory and contain no malignant cells, though at operation this can never be assumed.

Lymphatic Spread. In pages 3–5 the extensive nature of the lymphatic connections of the stomach has been emphasized. Gastric carcinomata frequently metastasize through the lymphatic system and this fact has to be taken into account in planning radical surgery. For the most part the direction of lymphatic spread is in accordance with the general plan of drainage shown in Fig. 7, but some important variations occur.

Growths of the upper third of the stomach and the cardia tend to involve all three lymphatic zones and, as Allison and Borrie (1949) and Eker (1951) have convincingly demonstrated by dissection of operative specimens, may therefore spread by all available lymphatic routes—to the glands around the cardia and accompanying the left gastric artery, to the pancreatico-lienal glands along the short gastric arteries and the splenic artery and pancreas, and to the glands along the gastro-epiploic arcade. It is to be noted especially that glands in the distant supra- and sub-pyloric regions may also be implicated.

The very much commoner growths of the pyloric region and distal half of the stomach, involving as they do only the lymphatic zones A and B, spread primarily to the glands on the gastric and gastro-epiploic arcades along the lesser and greater curves respectively. The gastro-epiploic glands are most numerous below the pyloric antrum—the sub-pyloric glands, which frequently contain metastases from pyloric carcinomata. Even more frequent is involvement of the lesser curve glands. From them spread may occur to the right to glands on the hepatic artery even as far as the portal fissure, but more commonly takes place to the left and upwards along the left gastric artery and eventually to glands on its main stem and around the cœliac axis. A sub-group of the left gastric glands is the chain surrounding the cardia, and these may be involved with the left gastric group even from a pyloric carcinoma—a significant point in regard to the prospect of eradicating such growths by a subtotal gastrectomy dividing the stomach to the right of the cardia. Further, as Eker (1951) has shown, carcinomata in the pyloric segment of the stomach may sometimes produce metastases in the glands of the pancreatico-lienal group.

Whatever glands may be affected primarily, the ultimate spread is to the cœliac glands, and in advanced growths there may be a solid mass of neoplastic tissue in the region of the cœliac artery. Further extension may occur from invasion of the nearby thoracic duct or other lymphatic channels draining upwards through the posterior mediastinum to the glands in the root of the neck, especially on the left side—*the glands of Virchow or Troisier* (1889). Alternatively extension may follow the Ligamentum Teres to the umbilicus and result in the development of an umbilical metastasis.

with a shallow irregular crater lined by necrotic malignant tissue, the edges being hard, raised and nodular. The base of the ulcer and the surrounding gastric wall may be much indurated. The serosa may be relatively normal in the early stages but later is usually invaded by the growth and is hard, white and nodular. When the part of the stomach containing the ulcer is cut across it is seen that the entire ulcer is raised above the surrounding mucosa and really projects into the lumen of the stomach, this being in sharp contrast with what obtains in simple peptic ulcer (*see* Fig. 19). Sometimes however an ulcerating carcinoma may closely resemble a benign peptic ulcer except that at one point on the circumference of the lesion more typical appearances of a new growth are seen in the form of a raised nodular edge or infiltration of the gastric wall.

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FIG. 20 Radiograph after a Barium meal showing an irregular filling defect due to a carcinoma of the pyloric region.

(Reproduced by courtesy of Dr E Rohan Williams)



FIG. 21. Radiograph after a Barium meal in a patient with pyloric obstruction due to carcinoma.

(Reproduced by courtesy of Dr. E Rohan Williams)



FIG 22 Radiograph after a Barium meal showing the radiological appearances of a "leather-bottle" stomach

(Reproduced by courtesy of Dr E Rohan Williams)



FIG. 23 Radiograph after an opaque meal showing a large malignant ulcer on the lesser curve The raised edges of the crater have caused a sharp indentation at the neck of the ulcer giving a partial meniscus sign

(Reproduced by courtesy of Dr. E Rohan Williams)

Blood Spread. At any stage in the development of the primary growth invasion of a vein in the gastric wall may occur and this may be followed by embolism of malignant cells in the portal vein, with the formation of hepatic metastases. These may involve one or both lobes, and may occur on the surface and be easily palpable, or be confined to the interior of the liver. (A solitary small secondary deposit on the surface of the liver may be extremely difficult to distinguish from a small cyst or angioma, at any rate on palpation.)

From the liver spread may occur via the blood stream to the lungs or rarely to the systemic structures—bones, skin, etc. These are usually late manifestations of the disease.

Clinical Features

Symptoms. The first symptoms are failure of appetite, loss of weight, and lack of energy, and the patient becomes anæmic. If the growth is situated in the middle part of the stomach well away from either orifice dyspeptic symptoms may be completely absent and the patient may attribute his complaint to overwork or post-influenzal debility, or detection of the anæmia may lead to a tentative diagnosis of pernicious anæmia. Though the anæmia in these cases is of secondary type it is associated with a fairly high colour index so that it may closely mimic a true Addisonian anæmia.

If the growth involves the pylorus there may be a vague indigestion consisting chiefly of fullness, pain and discomfort after meals. Usually the pain is slight and lacks the definition and regularity of a peptic ulcer pain, but sometimes in an ulcerating growth or cancer-ex-ulcere the pain may closely resemble that of a peptic ulcer. Eventually in these cases with pyloric lesions vomiting occurs and may be profuse; the vomitus often contains altered blood and is particularly offensive. Rarely does a patient with a gastric carcinoma suffer from a sudden severe hæmorrhage, necessitating his urgent admission to hospital. *With growths involving the cardiac orifice* the main symptom is progressive dysphagia as with carcinoma of the œsophagus.

In patients with a pre-existing chronic peptic ulcer the occurrence of malignant change is indicated by a striking alteration in the symptomatology. The pain loses its periodicity and is no longer relieved by meals and alkalis, the previous good appetite is replaced by distaste for food. In these cases free HCl is not only present in the gastric contents but may be excessive in amount.

Physical Signs. In early cases of carcinoma of the stomach there are no abnormal physical signs on clinical examination. Unfortunately most patients present late in the course of the disease and often manifest obvious abnormalities. There is usually evidence of loss of weight A palpable mass may be detected in the epigastric region with carcinomata of the pylorus or body of the stomach, or if there is gross pyloric obstruction gastric splashing may be elicited. The liver may be palpable, hard, and nodular due to metastases; sometimes jaundice is present from the same cause or from obstruction of the common bile duct by enlarged glands in the portal fissure. In cases with peritoneal deposits ascites may be demonstrable or a shelf of growth may be palpable in the recto-vesical or recto-uterine pouch on pelvic examination. Malignant invasion of the umbilicus may lead to its induration and fixation. The neck should always be examined for enlarged glands in the left or right supraclavicular region.

growth, but occasionally persistent clinical suspicion in the face of negative radiological and gastroscopic findings may necessitate exploratory laparotomy. In dealing with ulcerating lesions doubt as to whether the condition is benign or malignant sometimes remains after the stomach has been explored at laparotomy. In these cases it will be wisest to treat the ulcer as malignant unless it appears that by so doing the immediate operative risks will be greatly increased—as when a total gastrectomy replaces a partial gastrectomy—and the chances of the lesion being malignant are not held to be sufficiently great to justify these additional hazards.

Presymptomatic Diagnosis. The ideal obviously would be to diagnose gastric cancer, not merely soon after the onset of symptoms, but at a pre-symptomatic stage. Attempts to do so are being made in America in various Cancer Detection Clinics, such as at the Memorial Hospital for Cancer and Allied Diseases in New York. These cater for symptomless individuals usually over 45 or 50 years of age, who attend regularly for a complete physical examination and special investigations to exclude a malignant or premalignant lesion in any part of the body. So far as gastric cancer is concerned the essence of the method is radiological examination. A specially rapid technique is used to enable many patients to be X-rayed in a short space of time. Even so the radiological labour is considerable and the returns are apparently very small. St. John, Swenson and Harvey (1944) discovered only three carcinomata in 2,400 patients of over 45 years of age who were examined in this way. In some clinics to reduce the strain on the radiology department routine X-ray examination is replaced by gastric analysis; only those individuals with an achlorhydria or marked hypochlorhydria proceed to radiology Certain patients with gastric carcinoma unassociated with anacidity would of course escape diagnosis by this routine but the majority would be detected, and the scheme is more practicable than that of universal radiological investigation.

Complications of Carcinoma of the Stomach

(1) **Pyloric or Cardiac Obstruction.** This is frequently an integral part of the disease.

(2) **Hæmorrhage.** Usually this is of a chronic nature, but sometimes severe hæmatemesis and melæna are produced. This should be managed along the same line as hæmorrhage from a peptic ulcer, in most cases the bleeding ceasing under conservative treatment.

(3) **Perforation.** A gastric carcinoma, especially one arising in a pre-existing simple ulcer, may perforate into perigastric adhesions causing a localized abscess, or into the general peritoneal cavity. If free hydrochloric acid is present in the gastric secretion the perforation produces the same symptoms as does perforation of a peptic ulcer, but if there is complete achlorhydria the perforation may be much more insidious and give rise to peritonitis without any dramatic onset. The treatment consists of immediate operation at which the diagnosis of perforation is confirmed and the presence of a gastric carcinoma as the cause of the perforation is usually established. Sometimes the true nature of the initial gastric lesion may not be appreciated. Indeed it has been found by Doll (1950) that 8 per cent of a series of cases with perforations of what appeared to be simple gastric ulcers, eventually developed signs of carcinoma of the stomach, which was presumably responsible for their previous perforation. He has advocated immediate partial gastrectomy for all gastric—as contrasted with duodenal—perforations. Certainly a biopsy should be taken from the edge of the perforation in all cases

Special Investigations

RADIOLOGICAL EXAMINATION

This is the most valuable method of diagnosis, and it is useful even in cases which are clinically obvious, to show the precise situation and extent of the growth. An early growth may be revealed as a localized break in the mucosal relief pattern with some rigidity of the gastric wall. Well developed carcinomata may cause a large irregular filling defect (*see* Fig. 20) or may produce pyloric obstruction with gross dilatation of the stomach (*see* Fig 21). When the stomach wall is extensively infiltrated by carcinoma as in leather-bottle stomach it is shown radiologically to have rigid walls, a narrow lumen and to empty with unusual rapidity (*see* Fig. 22). The most difficult cases to diagnose radiologically are those with an ulcerating lesion on the lesser curve, because this may resemble a benign peptic ulcer. It has been said that any ulcer greater than 3 cm. across should be regarded as malignant, but this is not a reliable criterion because benign ulcers in this situation frequently exceed this size (*see* Fig. 16). In malignant lesions the ulcer niche is often less perfectly concave, there may be evident infiltration and induration of the adjoining gastric wall, if the ulcer is not too high up in the stomach for the radiologist to demonstrate this by suitable palpation, or the raised edges of growth may meet and shut off, partially or completely, the barium in the crater from that in the body of the stomach, as a discrete semilune or disc of opaque material—the meniscus sign of Carman (1913) (*see* Fig. 23). Carcinomata of the cardia may be revealed by a hold up of the swallowed barium at this point and by defective filling in this region.

GASTROSCOPY

See page 54

GASTRIC ANALYSIS

Usually a marked hypo- or achlorhydria is noted, but in a quarter of the cases the gastric acidity is within normal limits or excessive (Swynnerton and Truelove, 1952). Usually lactic or butyric acid is present due to fermentation. Exfoliated cancer cells have been demonstrated in the gastric contents on occasions, and methods of diagnosis have been developed which consist of rubbing off cells from the surface of the growth by means of a swallowed inflatable balloon with a roughened abrasive outer surface, from which the cells are subsequently recovered, stained and demonstrated microscopically.

EXAMINATION OF STOOLS FOR OCCULT BLOOD

Usually the presence of occult blood can be demonstrated by the benzidine test or by spectroscopy applied to the stools.

Diagnosis

Considerable delay often attends the diagnosis of cancer of the stomach due to the fact that the initial symptoms may be dismissed as being due to simple indigestion or anæmia. It cannot be too strongly emphasized that any indigestion, anæmia or loss of weight and energy arising in a patient over middle age demands thorough investigation. It is rare nowadays with good radiology, supplemented if necessary by gastroscopy, to be unable to reach a definite decision regarding the presence or absence of a malignant

growth, but occasionally persistent clinical suspicion in the face of negative radiological and gastroscopic findings may necessitate exploratory laparotomy. In dealing with ulcerating lesions doubt as to whether the condition is benign or malignant sometimes remains after the stomach has been explored at laparotomy. In these cases it will be wisest to treat the ulcer as malignant unless it appears that by so doing the immediate operative risks will be greatly increased—as when a total gastrectomy replaces a partial gastrectomy—and the chances of the lesion being malignant are not held to be sufficiently great to justify these additional hazards.

Presymptomatic Diagnosis. The ideal obviously would be to diagnose gastric cancer, not merely soon after the onset of symptoms, but at a pre-symptomatic stage. Attempts to do so are being made in America in various Cancer Detection Clinics, such as at the Memorial Hospital for Cancer and Allied Diseases in New York. These cater for symptomless individuals usually over 45 or 50 years of age, who attend regularly for a complete physical examination and special investigations to exclude a malignant or premalignant lesion in any part of the body. So far as gastric cancer is concerned the essence of the method is radiological examination. A specially rapid technique is used to enable many patients to be X-rayed in a short space of time. Even so the radiological labour is considerable and the returns are apparently very small. St. John, Swenson and Harvey (1944) discovered only three carcinomata in 2,400 patients of over 45 years of age who were examined in this way. In some clinics to reduce the strain on the radiology department routine X-ray examination is replaced by gastric analysis; only those individuals with an achlorhydria or marked hypochlorhydria proceed to radiology. Certain patients with gastric carcinoma unassociated with anacidity would of course escape diagnosis by this routine but the majority would be detected, and the scheme is more practicable than that of universal radiological investigation.

Complications of Carcinoma of the Stomach

(1) **Pyloric or Cardiac Obstruction.** This is frequently an integral part of the disease.

(2) **Hæmorrhage.** Usually this is of a chronic nature, but sometimes severe hæmatemesis and melæna are produced. This should be managed along the same line as hæmorrhage from a peptic ulcer, in most cases the bleeding ceasing under conservative treatment.

(3) **Perforation.** A gastric carcinoma, especially one arising in a pre-existing simple ulcer, may perforate into perigastric adhesions causing a localized abscess, or into the general peritoneal cavity. If free hydrochloric acid is present in the gastric secretion the perforation produces the same symptoms as does perforation of a peptic ulcer, but if there is complete achlorhydria the perforation may be much more insidious and give rise to peritonitis without any dramatic onset. The treatment consists of immediate operation at which the diagnosis of perforation is confirmed and the presence of a gastric carcinoma as the cause of the perforation is usually established. Sometimes the true nature of the initial gastric lesion may not be appreciated. Indeed it has been found by Doll (1950) that 8 per cent of a series of cases with perforations of what appeared to be simple gastric ulcers, eventually developed signs of carcinoma of the stomach, which was presumably responsible for their previous perforation. He has advocated immediate partial gastrectomy for all gastric—as contrasted with duodenal—perforations. Certainly a biopsy should be taken from the edge of the perforation in all cases

of gastric perforation treated by simple suture. For perforation of an obvious gastric carcinoma suture is often a difficult operation owing to the friability of the infiltrated stomach wall, and immediate gastrectomy is preferable if the patient's general condition will permit it.

Treatment

Radical Surgery. The only treatment that offers any prospect of cure is surgical excision of the growth and its extensions. This entails a wide gastrectomy with removal of the related lymph glands, greater and lesser omenta, and possibly parts of neighbouring organs. The operation may take the following forms:

Distal Subtotal Gastrectomy

This is the procedure used by most surgeons for carcinomata of the pylorus or distal half of the stomach, which incidentally represent some two-thirds of all gastric neoplasms. (For technical details *see* pages 85 and 87).

Proximal Partial or Total Gastrectomy

These operations have usually been reserved for growths of the cardia and upper part of the stomach, which cannot be removed by a distal subtotal gastrectomy. As carcinomata in these situations frequently metastasize to lymphatic glands in the hilum of the spleen and along the splenic vessels, it is a logical extension of these operations to remove the spleen and tail and body of pancreas *en bloc* with the stomach, as urged by Allison and Borrie (1949), and this addition does not seem to increase the risks. An abdomino-thoracic approach is usually required for these higher gastric resections, but for some growths in the upper part of the body of the stomach, too high for a distal partial gastrectomy, it may be possible to carry out *total gastrectomy (combined with spleno-pancreatic resection) by the abdominal route alone.* There are perhaps some advantages in a purely abdominal total gastrectomy when that is technically feasible; shock is probably less than with an abdomino-thoracic operation, and if the œsophago-jejunal anastomosis should leak, as it not infrequently does, this may not be fatal if the abdomen alone has been opened and the anastomotic site has been well drained, whereas after an abdomino-thoracic operation it is practically always lethal. For accounts of the technique of these operations reference should be made to pages 100 and 101.

It has been suggested by Longmire (1947) that all gastric carcinomata should be treated by total gastrectomy. The argument for this step is that even growths in the pyloric region may produce metastases in the glands around and to the left of the cardia or in the splenic hilum whch cannot be removed by any form of distal subtotal gastrectomy. It is probable however that such wide lymphatic spread occurs only in very advanced growths which would carry a poor prognosis with any operation. The operative mortality of total gastrectomy in most published series is twice or thrice that of subtotal gastrectomy for malignant disease (*see* Berkson, Walters, Gray and Priestley 1952) so that the late salvage rate would have to be very substantially increased to compensate for this initial loss. Another important consideration is the much more severe disturbance of digestive function and nutrition that occurs after a total then a subtotal gastrectomy, and this may make the patient's life extremely uncomfortable for the first

six or twelve months. Unfortunately most patients who submit to radical surgery for gastric carcinoma fail to achieve a complete cure and the main benefit that they derive from operation is a temporary relief of their symptoms. Obviously in this important palliative respect total gastrectomy is a much less satisfactory procedure than subtotal gastrectomy. These considerations have induced most surgeons to retain subtotal distal gastric resection, despite its imperfections, as the operation of choice for the majority of cases of gastric cancer.

Results of Radical Surgery

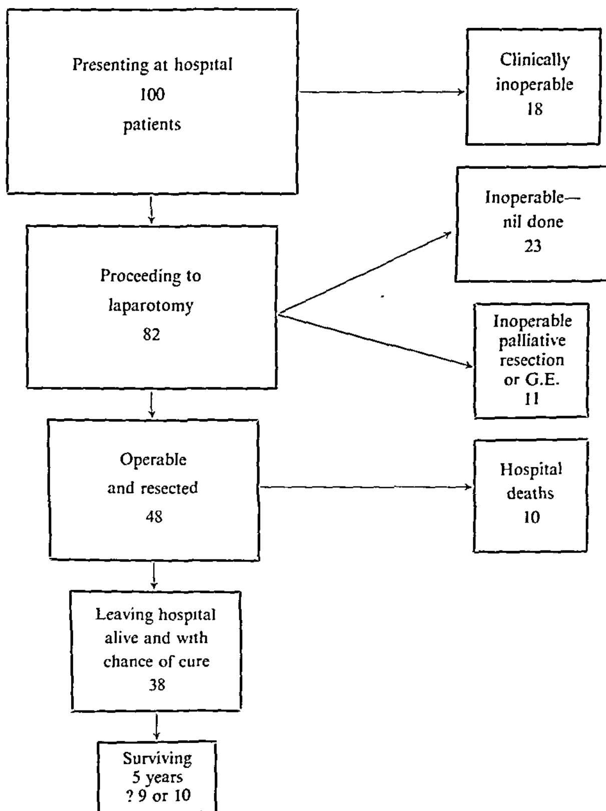
The achievements of radical surgery for carcinoma of the stomach can only be described as bitterly disappointing. Harnett (1947) reviewed the results of treatment of all patients presenting with carcinoma of the stomach at hospitals in the London area between April 1938 and September 1939. He found that of the 1,405 cases diagnosed as having malignant disease of the stomach only 245 proceeded to a radical operation, 163 survived that operation, and a *mere 36 or 2·56 per cent of the original* 1,405 *were alive and well five years later*. Since 1939 however the technique of total and proximal partial gastrectomy by the abdomino-thoracic approach has been developed and offers a chance of radical resection to patients with growths in the upper part of the stomach, which were previously quite inoperable. In addition there has been a remarkable increase in the safety of surgery in general due to a variety of factors, such as better methods of blood transfusion and anæsthesia, the introduction of antibiotics and a better understanding of the problem of fluid and electrolyte balance. As a consequence the surgeon can now do more for the sufferers from cancer of the stomach than was possible a decade ago. This is reflected in the results of treatment of gastric carcinoma at St. Mary's Hospital between 1947 and 1950 inclusive. In all, 134 cases were seen with the disease; the disposal and fate of these cases expressed on a percentage basis is shown in Table 1.

In most published reports on the late results of radical surgery for gastric cancer, a quarter or a fifth of the patients who survive operation live for at least five years subsequently (and in the group of cases without glandular metastases the proportion of the long-term survivors rises to 1 in 4 or 5). (*See* Harnett, 1947. Berkson, Walters, Gray and Priestley, 1952.) If a similar proportion of ultimate survivors obtains in this series, this would mean that some nine or ten of the 100 patients originally presenting for treatment would achieve five-year cures. This is certainly an improvement on the results recorded by Harnett but it still represents a small return for the expenditure of much surgical and nursing skill and effort. However it has to be remembered that even though many of these patients die of recurrence of their growths, they may often live in comfort for two years or more before they eventually succumb. After the removal of the primary growth their symptoms disappear and they usually improve enormously in general health, losing their cachectic appearance and putting on weight. Their subsequent recurrence may be in the liver and may cause much less discomfort than the initial growth. Radical surgery therefore has an unconscious palliative value which must not be disregarded in assessing its benefits.

Palliative Procedures. The greatest tragedy about carcinoma of the stomach is that even at the present day, by the time patients suffering from this disease reach hospital, some 50 per cent of them are already clearly beyond hope of cure by surgery. In some cases this is evident on clinical examination from the presence of ascites, a nodularly

TABLE I

Fate of 134 Patients with Gastric Carcinoma at St. Mary's Hospital from 1947 to 1950 inclusive, expressed on a Percentage Basis



enlarged liver, jaundice, pelvic deposits, deposits in the umbilicus or subcutaneous tissues of the abdominal wall, malignant glands in the neck or axillæ, or bony metastases. Sometimes a large fixed epigastric mass may indicate that the growth is probably irremovable, but the statement frequently made that a palpable tumour is always inoperable is certainly not true. Very rarely is operation contra-indicated on account of the patient's poor general condition from advanced age or concomitant disease. In the absence of a clear contra-indication, exploratory laparotomy should always be performed. This may reveal unsuspected hepatic or peritoneal deposits or local fixation and extension of the primary growth placing it beyond the reach of radical excision. Under these circumstances the possibility of a palliative operation should be considered.

Undoubtedly the most effective procedure is a *palliative resection* of the primary growth. This is performed in roughly the same manner as a resection for cure, but it need not be so extensive in dealing with related lymphatics. For cases with hepatic deposits or widespread lymphatic metastases excision is always worth doing if the primary lesion is removable and not more than half of the liver substance is adjudged to be replaced by growth. If the growth is too fixed, however, and pyloric obstruction is occurring or likely to develop in the near future, a *simple gastro-enterostomy* may be established, and is better done as an anterior than a posterior operation, because the anastomosis in the former is less liable to be occluded by subsequent spread of the growth. The symptomatic relief afforded by this procedure is often disappointingly small. A slightly more effective form of gastro-enterostomy is that known as *Devine's operation*. In this procedure the stomach is divided across proximal to the growth, the distal cross section closed, and the proximal one implanted in the side of the uppermost loop of jejunum (*see* Devine, 1928). It may be quite a difficult operation to perform. For patients with irremovable carcinomata of the cardia or fundus causing dysphagia, a *short-circuiting œsophago-jejunostomy* (*see* Volume Two) is a valuable procedure because it restores to the patient normal swallowing for his remaining few weeks or months of life. It is infinitely preferable to a gastrostomy. For cases with peritoneal deposits and ascites the short period of survival before them makes a palliative operation unnecessary as a rule.

In the later stages of inoperable or recurrent cases sedatives will have to be administered in increasing dosage. The effects of pyloric obstruction can be mitigated to some extent by regular gastric lavage. Radiotherapy is usually unavailing and may make the patients more uncomfortable.

CONGENITAL DUODENAL ATRESIA

This is an extremely rare condition where there is a failure of canalization of the endodermal rudiment of the duodenum. Usually the defect is situated just below the ampulla of Vater. It may vary in degree from the complete absence of a short segment of the gut to the formation of a partial or complete fibrous septum. A complete obstruction will lead to constant vomiting of bile-stained fluid, the vomiting beginning soon after birth and, if untreated, terminating in death. When an incomplete septum is present symptoms of chronic duodenal obstruction develop, namely gastric distension and periodic bilious vomiting. Clinically the condition may resemble a congenital hypertrophic pyloric stenosis but is distinguished by the earlier onset of symptoms and by the fact that the vomitus contains bile. A radiogram after an opaque meal will

usually clarify the diagnosis. An anastomosis between the duodenum above the obstruction and the upper jejunum gives complete relief of the obstruction; gastro-jejunostomy is a less effective procedure.

CHRONIC DUODENAL ILEUS

By this term is meant a chronic obstruction or dilatation of the duodenum. Two forms of the condition are recognized:

Duodenal Ileus due to an Obvious Cause

Here a definite obstructing lesion is present, such as calcified tuberculous mesenteric glands, adhesions or malignant infiltration of the upper part of the root of the small gut mesentery due to carcinoma of the stomach or intestine. The symptoms and signs of *this variety may resemble those of the second group or may be mainly those of the* causal condition. The treatment consists in removing the obstruction if that is possible, as for example by dividing adhesions, or, alternatively and more usually, by the performance of a short-circuiting lateral anastomosis between the distended duodenum above the obstruction and an upper collapsed loop of jejunum.

Duodenal Ileus without Obvious Cause

(So-called Arterio-Mesenteric Ileus)

This form occurs nearly always in visceroptotic middle-aged women who complain of attacks of bilious vomiting associated with nausea and a feeling of upper abdominal distension. As these patients are often introspective individuals, many other more or less vague symptoms may also be present. Radiological examination after an opaque meal will show considerable dilatation of the second and third parts of the duodenum, which usually terminates at the crossing of the superior mesenteric vessels.

It used to be believed on the basis of findings at laparotomy and at necropsy that the condition is caused by an unusual drag on the superior mesenteric vessels due to visceroptosis of the small gut, the result being that the third part of the duodenum was compressed between these vessels and the abdominal aorta. Sometimes ptosis of the cæcum and right colon with resulting tautness of the right colic artery as it crossed the duodenum appeared to be the factor responsible. In some cases however the dilatation of the duodenum is not sharply delineated distally by the crossing of the vessels mentioned, and indeed may not even reach beyond the ampulla of Vater, so that the theory of arterio-mesenteric compression is not applicable. In not a few cases the unusual distension of the duodenum may be due to sagging of the entire duodenal loop and part of a general visceroptosis.

Treatment. The treatment of duodenal ileus during an attack of vomiting is gastric lavage, gastroduodenal suction through an indwelling nasal Ryle's tube, the prone position with elevation of the foot of the bed on blocks, and intravenous fluids and electrolytes. Attempts are usually made to prevent further attacks by prescribing an abdominal support for the visceroptosis, though it is extremely doubtful if the position of abdominal viscera is ever much influenced by such supports. Finally, if symptoms become frequent and severe, operative treatment may have to be considered. This should take the form of a duodeno-jejunostomy with anastomosis between the side of the third part of the duodenum (rarely the second part if the dilatation does not extend

beyond it) and the uppermost loop of jejunum. Dramatic successes were claimed for surgery by Wilkie (1921) who first drew attention to the condition in the British literature, but many other surgeons have been less impressed with the results. Often the patient's symptoms continue little affected and one is driven to the conclusion that they are frequently manifestations of neurosis. The temptation to advise surgical intervention should therefore be resisted unless the ileus is gross and the symptoms are really severe.

DUODENAL DIVERTICULA

Diverticula of the duodenum are found in about 1 per cent of all patients undergoing radiological examination of the stomach and duodenum. They may be divided into two groups, primary and secondary diverticula.

Primary diverticula are usually found in the second portion of the duodenum, rarely in the third and never in the first. They appear as small flask shaped protrusions of the mucosa and submucosa through the muscularis of the duodenal wall, invariably on the concave side of the duodenal loop. They are thus retroperitoneal and closely related to the vessels entering the duodenum and to the head of the pancreas; they lie in front of, behind, or in the substance of the latter, and sometimes contain small islets of pancreatic tissue in their walls. Usually they are multiple.

Clinically these diverticula are usually quite symptomless and any complaint which the patient may have is nearly always referable to some other organic condition or to a psychoneurosis. The surgeon should be reluctant therefore to undertake surgical removal of a duodenal diverticulum, especially as the operation may be one of some intricacy. Very rarely do complications such as infection, perforation or carcinoma formation occur and require surgical treatment.

Secondary diverticula occur immediately proximal to stenosing duodenal ulcers and are therefore usually found in the first part of the duodenum. They are known as *pre-stenotic diverticula*. Their symptoms and treatment are those of the associated ulcer.

DUODENAL FISTULÆ

Internal fistula between the duodenum and neighbouring viscera, such as the gall bladder, small intestine or colon, may result from acute or chronic cholecystitis or the penetration of a chronic duodenal ulcer. It is also produced deliberately in the operation of cholecystoduodenostomy to short-circuit an irremovable obstruction of the common bile duct.

External fistula is much more common and important. It may be caused by accidental injury to the duodenum during right nephrectomy, right hemicolectomy or operations on the biliary tract. It may also follow inadequate closure of a perforated duodenal ulcer. But undoubtedly the commonest cause of an external duodenal fistula is leakage from the duodenal stump after gastrectomy, either from the blind end after resections of Polya type or from the gastroduodenal anastomosis after Billroth I operations. An external fistula usually shows itself within 7–10 days of the operation as an escape of bile-stained fluid from the wound. The subsequent course varies enormously from case to case. In some patients the discharge dries up in a very few days. In others it persists and becomes more profuse. As the fluid contains digestive ferments it digests the surrounding skin, which becomes red and raw, and the discomfort of the patient

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in the third part of the duodenum as typical malignant intestinal ulcers. *Secondary duodenal carcinomata* also may occasionally result from spread of growths of the stomach, pancreas, gall bladder or colon.

Clinical Features. In ampullary growths the presenting symptom is usually a painless, insidious obstructive jaundice, and the investigations are as for carcinomata of the head of the pancreas (*see* Chapter IV, page 244). Duodenal growths above or below the ampulla lead to duodenal obstruction, the symptoms are thus those of a high intestinal obstruction, at first intermittent and partial, later complete. Despite profuse vomiting, there is no abdominal distension and usually no mass to be felt in the abdomen. The precise diagnosis rests on radiological examination after an opaque meal. It is important for the radiologist to remember that these lesions do occur and to devote as much attention in his examination to the second and third parts of the duodenum as to the first. If symptoms are intermittent the lesion is more likely to be detected if the patient is examined radiologically during a symptomatic phase.

Treatment. Carcinomata of the duodenum arising in the region of the ampulla are treated as ampullary growths (*see* Chapter IV, page 245). Supra-ampullary carcinomata if operable will call also for a resection of the duodenum and head of pancreas. Infra-ampullary growths may require a similar resection or if near the duodeno-jejunal junction may be treated by a simple segmental resection of the terminal duodenum with end-to-end duodeno-jejunostomy. In inoperable cases the duodenal obstruction may be relieved by palliative gastro-jejunostomy, and the jaundice by cholecystenterostomy.

GASTRIC OPERATIONS

Pre-Operative Care

Many patients with uncomplicated peptic ulcer requiring operation are in excellent general health and need little pre-operative treatment Gross dental sepsis is perhaps better eliminated but this should be done several weeks beforehand. The patient is admitted 48 hours prior to operation and during this period should be instructed in the breathing exercises that will be an essential feature of his post-operative care. The hæmoglobin level and blood group are determined and one or two pints of blood are cross-matched in case of need during or after operation. A soap and water enema is given the evening before operation, and up to that time a normal light diet may be taken. Immediately before the patient comes to the theatre a Ryle's tube is passed and retained, and the stomach is emptied of any remaining contents by suction

Unfortunately other patients with peptic ulcer, or, more commonly, carcinoma, are found to be in poor general condition, due to vomiting, bleeding, and malnutrition. They may show gross anæmia and serious derangement of metabolism. In these cases a period of two or three weeks devoted to rectifying this state of affairs before operation is undertaken may be of inestimable value and may well determine the ultimate outcome. An accurate assessment of the extent of their anæmia, hypoproteinæmia dehydration, and depletion of electrolytes is the first essential. In addition to clinical examination their hæmoglobin level, red cell count, hæmatocrit-reading and plasma content of sodium, potassium and chloride ions and of protein should be determined. It is also helpful in planning treatment to know not only the quantity of the vomitus but also the concentration of the contained electrolytes.

on that account may be considerable. In addition, owing to the loss of fluid and electrolytes, the general condition may decline rapidly and the patient become dehydrated, depleted in electrolytes, especially sodium and potassium, and emaciated.

Treatment. External duodenal fistulæ should be treated conservatively in the first instance, and, though treatment may have to be prolonged, most of them will be found to close spontaneously in time. The essentials of a conservative regime are to diminish the amount of leakage as much as possible, to prevent digestion of the skin, and to replace the fluid and electrolytes that have been lost from the fistula. Stopping of mouth feeding and the institution of gastric aspiration through a Ryle's tube may be tried to see if the fistulous leakage is lessened, but frequently it is unaffected by these measures. To avoid skin digestion a rubber catheter may be placed in the fistula and connected to a suction apparatus to remove the fluid before it reaches the skin surface. Alternatively an adhesive ileostomy bag may be applied to restrict the area of exposed skin to a small circle immediately around the fistulous opening, or the patient may be nursed in a prone position so that the fluid drops directly from the fistula into a kidney dish without running over the anterior abdominal wall. The fluid lost from the fistula is measured and its electrolyte content determined so that accurate replacement may be effected intravenously. In addition, if mouth feeding has been stopped, the normal basic requirement of fluid and electrolytes will have to be given parenterally. An effort may be made to increase the calorie content of the intravenous infusion by including 10 per cent invert sugar or alcohol. If after 10 or 14 days the fistula shows no signs of closing a jejunostomy may be established, partly for feeding purposes by means of a tube passed distally, and partly for aspiration of the duodenal contents by another tube passed proximally.

Most supra-ampullary fistulæ heal either with expectant treatment or with the conservative surgical measures just described. Infra-ampullary fistulæ, such as may result from a side injury of the lower part of the duodenum during a hemicolectomy, usually continue discharging till operative closure is carried out. Unless therefore they soon show signs of spontaneous improvement they should be dealt with by direct surgical attack.

TUMOURS OF THE DUODENUM

Simple Tumours

These are excessively rare, but benign tumours of the same varieties as those seen in the stomach have nearly all been encountered in the duodenum.

Carcinoma

Unlike the stomach the duodenum is very seldom affected by carcinoma; and, by contrast with gastric ulcers, chronic duodenal ulcers practically never become malignant. But inch for inch the duodenum is more likely to undergo malignant degeneration than is any other part of the small intestine Duodenal carcinomata are said to represent some 0·3 per cent of all carcinomata of the gastro-intestinal tract (Rogers, Goligher and Williams, 1952).

The commonest site for a carcinoma in the duodenum is in the vicinity of the ampulla of Vater, and it is often difficult to decide whether a growth in this situation has originated in the duodenal mucosa or in the ampulla itself. Probably most of them are primarily ampullary, but true duodenal carcinomata do occur in this situation and also

discharged to suitable convalescent accommodation. During convalescence there is no need to adhere to any special diet as a rule, but, after gastrectomy, patients usually find that they cannot manage full-sized meals and prefer to supplement their main meals with large snacks at 11 a.m. and at 4 p.m. Some surgeons advise alkalis for duodenal ulcer patients for a time after operation but, if the operation has achieved its objective of controlling hyperchlorhydria, this should be quite unnecessary.

Anæsthesia

Formerly spinal anæsthesia, or local and splanchnic anæsthesia, were popular with many surgeons for gastric work, but the great success and safety of modern general anæsthesia supplemented by relaxant drugs has now practically eliminated these methods. If the spontaneous heavy respiration of deep general anæsthesia is completely suppressed by increasing the dosage of the relaxant agent, and quiet artificial respiration by compression of the anæsthetic bag is substituted, operative manœuvres immediately below the diaphragm are greatly facilitated, and the operating conditions for the surgeon are well-nigh perfect. In the writer's opinion there remains only one possible indication for avoiding general anæsthesia and that is in operating on a patient with hæmatemesis. The risk of vomiting fluid and clotted blood during induction of anæsthesia and their subsequent inhalation into the respiratory tract is very real in this type of case and requires the utmost vigilance and celerity on the part of the anæsthetist. If an experienced anæsthetist is not available it will be safer to rely on a local and splanchnic block which leaves the patient conscious and able to cope safely with his own vomitus.

For local anæsthesia a dilute solution of amethocaine 1/2,000 in saline, with adrenaline hydrochloride added to a concentration of 1/400,000, is the one preferred by the writer. It is injected into the musculature of the abdominal wall along the outer edges of the recti and along the costal margin to the xyphoid cartilage. Some solution is also injected into the subcutaneous tissues in the line of the proposed incision. Up to a total of 250 ml. may be used. After the abdomen has been opened the liver is retracted upwards and the intervertebral disc between the twelfth Th. and first L. vertebræ is palpated. A special long Finsterer needle is inserted through the posterior peritoneum at this level and between the abdominal aorta and the inferior vena cava; if no blood escapes it is connected to a syringe and 60–80 ml. of the same amethocaine solution are injected and diffuse into the region of the cœliac plexus. Anæsthesia is usually complete in 7–10 minutes.

Partial Gastrectomy

Two forms of distal partial gastrectomy are in common use. They derive from the two techniques of gastric resection originally worked out for the treatment of pyloric carcinoma by Theodor Billroth in Vienna between 1881 and 1885, and appropriately designated Billroth's first and second methods respectively (*see* Wölfler 1881, Hacker 1885, and Brunschwig 1951). Actually Billroth was preceded in his performance of the first technique by J. Péan of Paris who made an unsuccessful attempt to remove a gastric cancer in 1879. But it was the work of Billroth and his numerous assistants that established these operations and it is fitting that this should be recognized eponymously.

The original Billroth I operation consisted in resecting the pyloric segment, and joining the remainder of the stomach to the duodenum by end-to-end anastomosis. It

Dehydration and electrolyte imbalance should be corrected by intravenous administration of appropriate amounts of dextrose water, normal saline, or ammonium or potassium chloride solution, the effect being gauged by further estimations of plasma electrolytes. Anæmia may require one or more pre-operative blood transfusions. Protein depletion is best combated by a high protein, high calorie diet for a period of two or three weeks, but if pyloric obstruction interferes with natural feeding then aminoacid mixtures such as Casydrol may be given intravenously. However unless the calorie content of the infusion can be raised to 3,000 per day, the aminoacids are utilized not for tissue repair but to provide further calories. (The problem of achieving a really high calorie intake in the infusing fluid has only been partly solved. It is usually met by giving a mixture of invert sugar solution and ethylalcohol, but this is liable to result in early venous thrombosis; in the future satisfactory emulsions of fat for intravenous administration may become available.) Large doses of vitamins B and C should also be given to these patients. If obstruction is present gastric lavage should be performed twice daily for several days before operation. Some surgeons administer hydrochloric acid by mouth to achlorhydric patients with gastric carcinomata to aid in the sterilization of the gastric contents.

Post-Operative Care

On recovering from the anæsthetic the patient is most comfortably nursed in the semi-reclining position. Respiratory exercises and exercises for the lower limbs are commenced that evening if possible and are continued twice daily for a week. The patient usually gets out of bed and takes a few steps the evening of the day after operation, and his activities are gradually increased. Mouth feeding of small quantities of water can be commenced as soon as consciousness returns and these feeds are gradually increased from 1 oz. in every hour on the first day to 3 or 4 oz. hourly of a mixture of milk and water on the third or fourth day, provided that aspiration through the Ryle's tube every hour shows that there is no accumulation in the stomach likely to stretch the suture lines. At this stage the Ryle's tube may be withdrawn and the feeds augmented with Benger's food, jelly, chicken tea. By the end of the first week the patient is taking a light diet of switched eggs, milk puddings, bread and butter, and minced chicken, with feeds of milk in between the main meals.

During the first two or three post-operative days the allowance of fluid by mouth on this regime is clearly insufficient to satisfy the patient's needs and additional fluid must be given parenterally. A total intake of some three litres is usually desirable and in addition fluid aspirated from the stomach has to be made good. The basic parenteral fluid should consist of N/5 normal saline; gastric aspirations however should be compensated for by administration of normal saline or if possible the precise ionic content of the aspirate should be determined and the composition of the replacing fluid for the next 24 hours chosen accordingly. An accurate record should be kept of the fluid and electrolyte balance during the initial few days at any rate, till all parenteral feeding has ceased. During this early period of essentially parenteral feeding, strict attention to oral hygiene is essential, and the mouth should be washed and swabbed out four-hourly for three or four days. Penicillin or other antibiotic is given systematically as a routine for at least a week to minimize the risks of broncho-pulmonary infection.

The skin sutures are removed on the eighth to tenth day and the patient can then be

discharged to suitable convalescent accommodation. During convalescence there is no need to adhere to any special diet as a rule, but, after gastrectomy, patients usually find that they cannot manage full-sized meals and prefer to supplement their main meals with large snacks at 11 a.m. and at 4 p.m. Some surgeons advise alkalis for duodenal ulcer patients for a time after operation but, if the operation has achieved its objective of controlling hyperchlorhydria, this should be quite unnecessary.

Anæsthesia

Formerly spinal anæsthesia, or local and splanchnic anæsthesia, were popular with many surgeons for gastric work, but the great success and safety of modern general anæsthesia supplemented by relaxant drugs has now practically eliminated these methods. If the spontaneous heavy respiration of deep general anæsthesia is completely suppressed by increasing the dosage of the relaxant agent, and quiet artificial respiration by compression of the anæsthetic bag is substituted, operative manœuvres immediately below the diaphragm are greatly facilitated, and the operating conditions for the surgeon are well-nigh perfect. In the writer's opinion there remains only one possible indication for avoiding general anæsthesia and that is in operating on a patient with hæmatemesis. The risk of vomiting fluid and clotted blood during induction of anæsthesia and their subsequent inhalation into the respiratory tract is very real in this type of case and requires the utmost vigilance and celerity on the part of the anæsthetist. If an experienced anæsthetist is not available it will be safer to rely on a local and splanchnic block which leaves the patient conscious and able to cope safely with his own vomitus.

For local anæsthesia a dilute solution of amethocaine 1/2,000 in saline, with adrenaline hydrochloride added to a concentration of 1/400,000, is the one preferred by the writer. It is injected into the musculature of the abdominal wall along the outer edges of the recti and along the costal margin to the xyphoid cartilage. Some solution is also injected into the subcutaneous tissues in the line of the proposed incision. Up to a total of 250 ml. may be used. After the abdomen has been opened the liver is retracted upwards and the intervertebral disc between the twelfth Th. and first L. vertebræ is palpated. A special long Finsterer needle is inserted through the posterior peritoneum at this level and between the abdominal aorta and the inferior vena cava; if no blood escapes it is connected to a syringe and 60–80 ml. of the same amethocaine solution are injected and diffuse into the region of the cœliac plexus. Anæsthesia is usually complete in 7–10 minutes.

Partial Gastrectomy

Two forms of distal partial gastrectomy are in common use. They derive from the two techniques of gastric resection originally worked out for the treatment of pyloric carcinoma by Theodor Billroth in Vienna between 1881 and 1885, and appropriately designated Billroth's first and second methods respectively (*see* Wölfler 1881, Hacker 1885, and Brunschwig 1951). Actually Billroth was preceded in his performance of the first technique by J. Péan of Paris who made an unsuccessful attempt to remove a gastric cancer in 1879. But it was the work of Billroth and his numerous assistants that established these operations and it is fitting that this should be recognized eponymously.

The original Billroth I operation consisted in resecting the pyloric segment, and joining the remainder of the stomach to the duodenum by end-to-end anastomosis. It

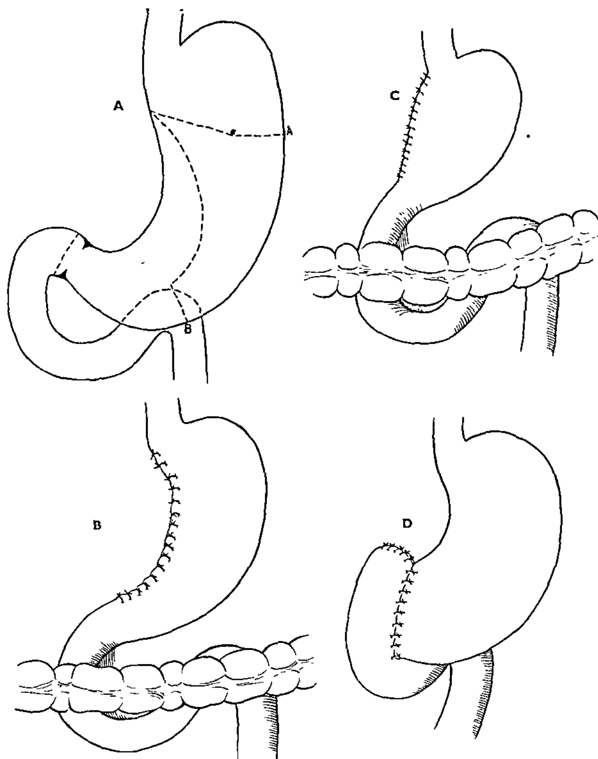


FIG 24 Diagram showing some of the commoner variants of the Billroth I resection

(a) Lines of section indicated on the intact stomach "A" for modern Billroth I operation, "B" for Shoemaker operation
(b) Shoemaker gastrectomy
(c) Modern *Billroth I gastrectomy*
(d) Haberer-Finney gastrectomy

was found that leakage occasionally occurred from the angle between the lesser curve and the superior border of the duodenum, and this angle therefore came to be known as the "Jammerecke or angle of lament." Because of this complication the Billroth I technique fell temporarily out of favour but was subsequently revived by Continental surgeons, notably Shoemaker (1911) and von Haberer (1933), and more recently has been extensively used in this country. It has been found possible to perform a really high gastrectomy by this method and yet bring the stomach and duodenal stump together without undue tension or subsequent leakage if the upper two-thirds or three-quarters of the stomach is closed by suture and only the lowermost part used for the actual gastro-duodenal anastomosis. Shoemaker devised a special curved clamp to facilitate the closure of the upper part of the stomach along a line extending in an oblique curve from high on the lesser curvature of the stomach to a point low down in the body of the stomach, as in Fig. 24 (*a*) and (*b*), but most surgeons find that they can manage quite well with any reliable type of crushing clamp such as a large Payr clamp (*see* Fig. 24 (*a*) and (*c*)). In operating on cases of duodenal ulcer where the first part of the duodenum was difficult to prepare for anastomosis, von Haberer developed the technique of closing the duodenal stump and anastomosing the end of the stomach to the side of the second part of the duodenum, and this method was preferred also by Finney (1923) (*see* Fig. 24 (*d*)), but this modification of the Billroth I operation is seldom used at the present day.

In the original Billroth II operation the resection of the distal part of the stomach was followed by closure of the open ends of the duodenum and the gastric remnant, continuity of the alimentary tract being restored by establishing a side-to-side anastomosis between the front of the stomach stump and the uppermost loop of jejunum. But if the growth were large it was sometimes found difficult to carry out a sufficiently extensive resection and retain enough stomach to permit of the easy performance of an anterior gastro-enterostomy. Also it was wasteful of time to suture the open end of the stomach stump and then make a fresh opening on its anterior wall. Several surgeons

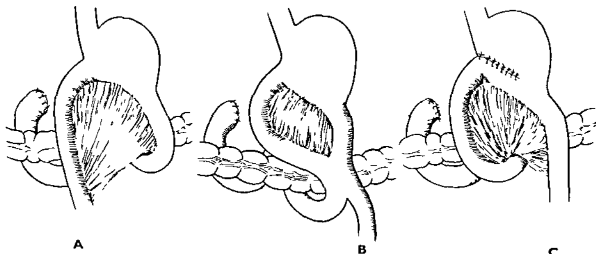


FIG 25 Diagram showing common antecolic variants of the Billroth II or Polya resection
(*a*) Anterior Polya-Moynihan operation
(*b*) Anterior Polya operation with entero-anastomosis
(*c*) Anterior Hofmeister-Finsterer operation with restricted stoma and valve.
It should be emphasized that many surgeons prefer similar retrocolic modifications of the Polya operation, which employ a shorter jejunal loop

therefore, including Krönlein (1888), preferred to implant the cut end of the stomach into the side of the jejunum, but this method did not become common practice till Eugen Polya of Budapest published his important paper on the subject in 1911. Numerous variations in the technique of the Billroth II or Polya operation have been advocated, the methods varying mainly according to the method of approximating the jejunum to the stomach stump and its relationship to the colon. Some commonly used techniques are indicated in Fig. 25 (*a*), (*b*), (*c*), and described in greater detail by Polya (1940). A popular method is that often associated with the names of Hofmeister (1896) and Finsterer (1914) on the Continent, and of Lake (1937) in this country, in which the upper part of the cross section of the stomach is closed and only the lower portion used for the actual anastomosis to the jejunum. Attaching the proximal jejunal loop to the closed upper section of the stump then creates a valve, which may have some value in preventing the entrance of gastric contents into the afferent loop of jejunum and duodenum (*see* Fig. 25 (*c*)).

Detailed Technique of Partial Gastrectomy. In the performance of a partial gastrectomy by any of the methods mentioned above the minutiæ of technique will vary from surgeon to surgeon. The following is a detailed account of the methods preferred by the writer, which are the anterior Hofmeister-Finsterer and Billroth I operations.

The Anterior Hofmeister-Finsterer Operation

The writer has used transverse epigastric incisions for gastrectomy particularly in rather obese patients with broad epigastria, and has found that they provide a reasonably good approach and leave a strong wound subsequently. For most cases however a vertical incision, either paramedian or median, gives better access to the upper part of the stomach. A right paramedian incision, displacing the rectus muscle, is generally preferred for duodenal ulcer cases, and a left paramedian for those with a high gastric ulcer. The incision extends up to the costal margin beside the xyphoid cartilage and down as far as the umbilicus. To obtain proper access to the upper part of the stomach through this approach it is necessary to divide the Ligamentum Teres and falciform ligament between forceps. The stomach and duodenum and other organs are then examined, the lesion is demonstrated and its fixity to other parts ascertained. If it should be a carcinoma the extent of spread of the growth must also be determined and a decision made as to operability.

The first step in the actual gastrectomy is to render the greater omentum taut by lifting up the middle portion of the stomach and the transverse colon and drawing them gently apart. Unless the patient is very obese it is usually possible to see through the thinner parts of the omentum into the lesser sac and to make a snick with scissors in one of the avascular areas. Starting at this small opening the greater omentum is divided progressively between forceps (*see* Fig. 26) towards the lower border of the first inch of the duodenum, the line of division running parallel to the greater curve of the stomach and usually below the gastro-epiploic arcade. As the division of the omentum proceeds it is important to ensure that the transverse mesocolon is not adherent to the posterior surface of the pyloric part of the stomach and to the omentum; if necessary it must be brushed off these structures, otherwise it may be inadvertently divided and the middle colic vessels damaged. As the lower border of the duodenum is approached the right gastro-epiploic or gastro-duodenal vessels are exposed and divided; in addition many

other small unnamed vessels have to be caught and severed before the duodenum is completely bared on this aspect. If the greater omentum is divided, as described, below the gastro-epiploic arcade, it is deprived of most of its blood supply and as a consequence subsequently undergoes considerable atrophy. However no harm seems to result, and this technique has the advantage of being less time-consuming than the method of division of the omentum between the arcade and the greater curve of the stomach advocated by Finsterer (1914).

The next step is to free the upper border of the duodenum by division of the right gastric artery and other small vessels descending from the hepatic artery in the lesser omentum to the duodenum. This is done by passing a finger up behind the duodenum and through the thin avascular part of the lesser omentum an inch or so above the duodenum and lesser curve. The tissue lying between the finger and the upper border of the duodenum contains these vessels; it should be underrun by dissecting forceps, inserted just above the duodenum, and divided between artery forceps. If there is much scarring extending into the lesser omentum at the upper border of the duodenum, the common bile duct and hepatic artery may be drawn close to the duodenum, and it will be safer to apply only one pair of forceps as close to the duodenal wall as possible and to divide between it and the duodenum. The other end of the artery will then have to be caught as it bleeds, but sometimes it has become obliterated by fibrosis. This is the best method of avoiding damage to the common bile duct in this situation and makes it unnecessary to expose the duct in the lesser omentum, as advised by some surgeons.

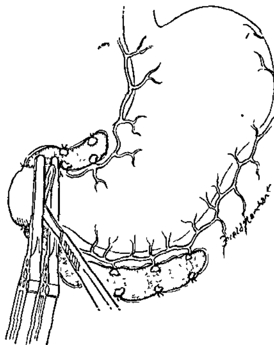


FIG. 26 Distal partial gastrectomy Greater and lesser omentum have been divided in pyloric region and crushing clamps applied to the duodenum preparatory to its division

In a case with a non-adherent duodenum it will be found that this organ is now sufficiently mobilized and bared to be divided between crushing clamps just beyond the pyloro-duodenal junction if operating for gastric ulcer, or immediately distal to the lesion itself if for duodenal ulcer. This leaves a sufficient duodenal stump in the grip of the more distal forceps for closure by either of the methods shown in Figs. 27 and 28, of which the writer prefers the latter.

If there is an adherent posterior wall duodenal ulcer however a decision must be made as to whether it is in fact resectable or whether it should be left and the resection terminated proximal to the ulcer

(*a*) RESECTION TO BEYOND THE ULCER. It is helpful in these cases to start by dividing any adhesions on the anterior aspect of the duodenum so as to define clearly its upper and lower borders. The posterior duodenal wall has then to be separated *carefully* by a combination of sharp and blunt dissection from the neck of the pancreas and the stem of the gastroduodenal artery, numerous small vessels running into it from these structures

being divided in the process. Sometimes the duodenum with its contained ulcer can be completely separated from the pancreas. When the ulcer penetrates deeply however, as the dissection proceeds, the duodenum is usually opened leaving the ulcer crater adherent to the pancreas. In these cases it is necessary to separate the posterior duodenal wall from the pancreas for another half inch to three-quarter of an inch distal to the ulcer in

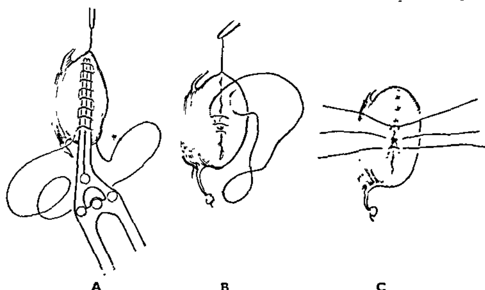


FIG. 27 Closure of duodenal stump by Moynihan-Mikulicz invagination mattress stitch applied over crushing clamp

(a) Insertion of running mattress stitch
(b) Clamp has been removed and mattress suture drawn tight securing invagination of edges, stitch being returned along stump producing further apposition of peritoneal surfaces
(c) Insertion of Lembert sutures

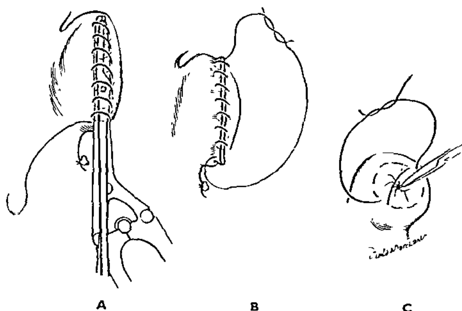


FIG. 28 Alternative method of [illegible]

Z-shaped Lembert suture —this may be repeated once or twice.

order to secure enough free duodenum for safe closure. The duodenal stump is then closed with a running catgut suture, preferably of Connell type, to invert the edges. This is followed by one or two layers of interrupted Lembert sutures of fine silk. Finally

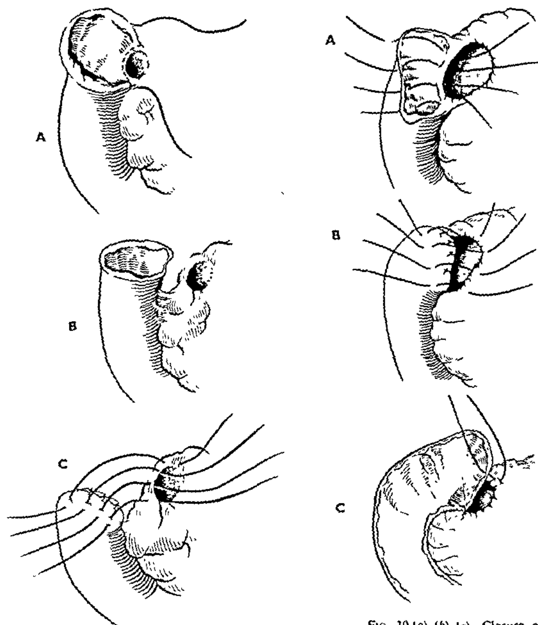


FIG 29 (*a*), (*b*), (*c*) Closure of duodenal stump by dissection beyond penetrating posterior wall ulcer

FIG 30 (*a*), (*b*), (*c*) Closure of duodenal stump by Nissen's method, suitable for cases with an adherent posterior wall duodenal ulcer

the closed stump may be stitched against the capsule of the adjacent pancreas or the ulcer crater, if this can be accomplished without tension (*see* Fig. 29 (*a*), (*b*), (*c*)).

If it is thought that dissection between the posterior duodenal wall and the pancreas beyond the ulceration may endanger the common bile duct, drawn out of its normal course by scarring, the upper edge of the posterior duodenal wall may be left attached to the distal border of the crater and the free edge of the anterior wall sutured on to the crater as recommended by Nissen (1945) (*see* Fig. 30 (*a*), (*b*), (*c*)).

(*b*) PREPYLORIC SECTION. If the ulcer is considered too large and fixed or situated too far distally in the duodenum to be resected with safety, it will have to be left in the duodenal stump. Under these circumstances it will usually be found necessary to retain a fringe of pyloric antrum attached to the duodenal stump in order to permit of its safe suture proximal to the ulcer. This method of prepyloric section through the antrum, or "resection for exclusion" was warmly recommended by Finsterer (1918) as a safe means of dealing with adherent duodenal ulcers, but it has been shown by Ogilvie (1947) to be particularly liable to be followed by stomal ulceration, presumably because continued production of gastrin by the mucosa of the retained portion of antrum leads to hypersecretion of acid by the gastric stump. Therefore if resection for exclusion is practised it is essential that all the antral mucosa should be "cored out" from the pyloro-duodenal stump down to the actual pyloroduodenal junction by the technique described by Plenk (1936) and Bancroft (1932) (*see* Fig. 31 (*a*), (*b*), (*c*)). Alternatively the mucosa may be left temporarily, and a second operation performed five or six weeks later to excise the remnant of antrum and the part of the duodenum containing the then healed ulcer as advocated by McKittrick and Moore (1945). The disadvantage—perhaps largely theoretical—of the latter method is that the patient who feels very well after the first operation may refuse to proceed to the second stage The frequency with which prepyloric section is deemed necessary in dealing with adherent duodenal ulcers varies according to the skill and experience of the surgeon. In his earlier days he may use it fairly often, but once he has mastered the technique of duodenal dissection he will practically always be able to resect to beyond the ulcer.

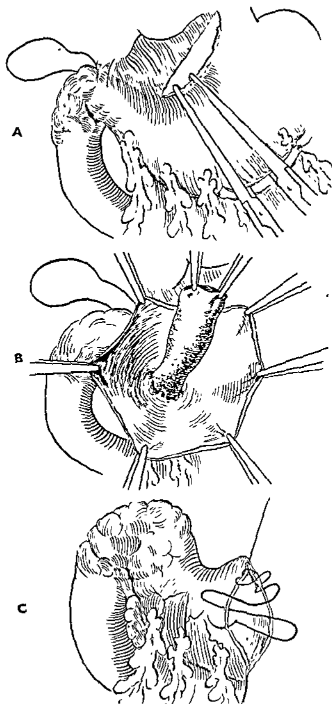


FIG. 31 (*a*), (*b*), (*c*) Bancroft or Plenk method of prepyloric section of the stomach with "coring out" of the antral mucosa

Attention is now turned to freeing the stomach and preparing it for resection. A useful preliminary

is to remove the clamp at its distal divided end and to aspirate the gastric contents completely; the collapsed and flaccid organ is then much easier to handle during the next phase of the operation. The clamp is thereafter reapplied and covered with a pack or a Maingot shield. The left edge of the wound is strongly retracted and the stomach and transverse colon are drawn to the right and somewhat apart to expose the intact part of the greater omentum. Forceps are applied to the latter in turn and the omentum divided between them up to the point on the greater curve selected as the proximal limit of the resection. In a subtotal gastrectomy for a duodenal ulcer it is usual to divide the left gastro-epiploic artery and two or three of the short

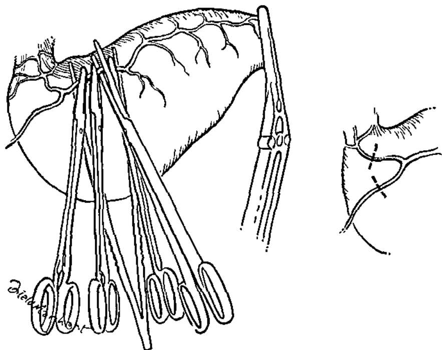


FIG 32. Clamping and dividing the left gastric vessels high on the lesser curvature by the "3 artery forceps" method Inset shows higher section involving division of the main stem of the left gastric and its œsophageal branches, employed in operating for high gastric ulcers or gastric carcinomata

gastric branches, but in a resection for a gastric ulcer much of the greater curve, all the vasa brevia, and possibly also the greater part of the left gastro-epiploic artery may be conserved.

The mobilization of the gastric stump is now completed by ligation and division of the left gastric vessels. The liver is retracted by a deep liver retractor and the stomach is lifted vertically upwards and to the left exposing the artery and its accompanying vein as it proceeds from the cœliac axis to the lesser curve of the stomach. In a subtotal gastrectomy for duodenal ulcer, or in operating for an ulcer high on the lesser curve, these vessels are best taken either just where the artery meets the lesser curve and sends its œsophageal branches upwards (*see* Fig. 32), or else at its origin from the axis, in which case the œsophageal branches will have to be divided separately at a higher level. In a lower resection the left gastric vessels are taken on the lesser curve itself at the point selected for division of the stomach. In ligating the left gastric at any of these levels, the procedure which the writer favours is to underrun it with a pair of large dissecting forceps.

Three pairs of artery forceps are then applied and the vessel is divided between them, leaving two forceps on the proximal end of the vessel. A strong linen thread ligature, No. 20, can be applied to the vessel in the groove made by the most proximal forcep whilst the middle forcep is still *in situ* to prevent slipping. The forceps on the gastric end of the vessel are used to strip the uppermost part of the lesser omentum downwards off the upper end of the lesser curve, any small branches going to the stomach divided between forceps in the process.

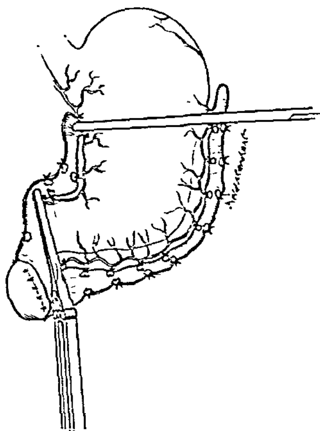


Fig. 33 Anterior Hofmeister-Finsterer gastrectomy Application of spring clamp to the stomach at superior limit of mobilization

The stomach is now ready to be divided and anastomosed to the jejunum. A spring clamp is applied one inch above the proposed line of section and the part of the stomach distal to this is turned upwards and to the left on to the left chest (*see* Figs. 33 and 34). A loop of jejunum is now brought up in front of the transverse colon, to be apposed to the stomach. The afferent limb is taken to the lesser curve and a piece of gut 8–10 in. long as measured from the duodenum-jejunal junction, is required to reach without tension and without undue constriction of the colon. A spring clamp is applied to the jejunal loop at the appropriate site and this clamp is brought alongside the clamp controlling the stomach. A moist swab is placed between them, packs are applied around to diminish contamination, and the anastomosis is commenced.

Using a series of interrupted fine silk or cotton Lembert sutures, or a continuous stitch of No. 2/0 chromicized catgut, the projecting jejunum and stomach are united throughout their length just distal to the spring clamps. A small Payr clamp is now applied to the lesser curve half or two-thirds of the stomach half an inch beyond the Lembert stitch. Beyond this again, across the whole stomach, a spring clamp is placed to prevent leakage whilst the stomach is divided between two clamps (*see* Fig. 35). A scalpel is used for the upper part of the section and it cuts on to the Payr clamp; for the lower half beyond the reach of the latter clamp the cutting diathermy or scissors are better. A running mattress stitch of No. 1/0 or 2/0 chromicized catgut is inserted with a straight needle immediately proximal to the Payr clamp, which is then removed. This stitch apposes the crushed edges and prevents protrusion of the mucosa but it does not control bleeding. For hæmostatic purposes it is essential to return the catgut suture along the closed edge of the stump as a closely placed over-and-over stitch. The stomach has now been closed except for the lower half or third of its cross section. An opening of corresponding size is now made in the jejunum using the cutting diathermy current (*see*

Fig. 36 (*a*)). The respective openings are then approximated by a continuous No. 2/0 chromic catgut through-and-through suture, which unites firstly the posterior edges and then the anterior borders (*see* Fig. 36 (*a*), (*b*), (*c*), (*d*)). If the anterior through-and-through stitch is inserted on the Connell principle to invert the mucosa, the individual vessels in the submucosa should be clipped with fine artery forceps and tied off with No. 2/0 catgut before the suture is begun.

The spring clamps and surrounding packs may now be removed from the stomach

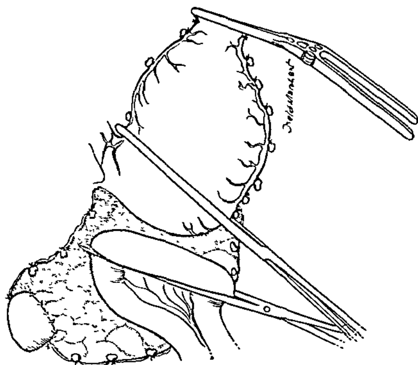


Fig. 34. Anterior Hofmeister-Finsterer gastrectomy Stomach thrown to the left exposing posterior surface distal to the occlusion clamp Approximation of upper loop of jejunum controlled by spring clamp and insertion of posterior row of interrupted Lembert sutures

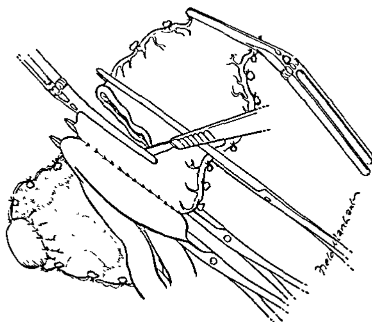


Fig. 35 Anterior Hofmeister-Finsterer gastrectomy. Closure of upper two-thirds of stomach cross-section by Payr clamp and excision of stomach distal to this.

and duodenum as the lumina of the stomach and jejunum have been securely closed. It remains simply to insert the anterior row of Lembert sutures of interrupted fine silk or continuous catgut to finish the anastomosis (*see* Fig. 36 (*d*)). As the afferent limb of jejunum attached to the lesser curve end of the gastric stump will tend to drag on this part of the anastomosis when the patient is in the sitting or erect position it is perhaps wise to reinforce the gastrojejunal approximation here by inserting two or three mattress sutures. A stitch or two between the afferent limb a short distance proximal to this point and the front of the pancreas, as recommended by Maingot (1948), may also take some of the strain off this part of the anastomosis and will prevent the afferent jejunal limb from rotating to the left and kinking.

Before closing the abdomen it is as well to inspect the region of the spleen, duodenal stump, right and left gastric pedicles to make sure that no bleeding is occurring from the

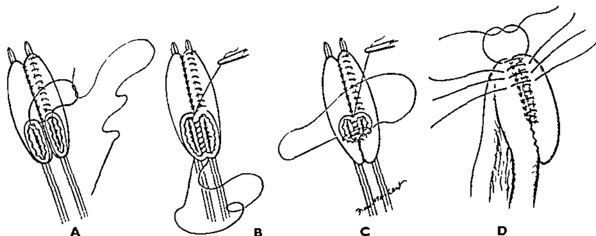


FIG 36 Anterior Hofmeister-Finsterer gastrectomy:
(*a*) Opening made in the lower part of the jejunal loop corresponding in size to the lower open part of the stomach
(*b*)
(*c*)
(*d*)

various ligated pedicles Some penicillin-sulphonamide powder is then applied to the vicinity of suture lines. A debatable point is whether or not a drain should be inserted down to the duodenal stump in cases where its closure has been difficult, as a safeguard in the event of leakage. It is argued by those who oppose this step that by causing irritation the drain may encourage the suture line in the stump to break down and leak. The writer however frequently uses a drain and believes that if it is to do any good it should be left *in situ* for at least six or seven days.

The wound is closed in layers, using a continuous No. 2 chromic catgut stitch for the posterior rectus sheath and peritoneum, and the same, or nylon, stainless steel wire, linen or silk, as interrupted or continuous sutures for the anterior rectus sheath. The skin edges are apposed by suture or by Michel clips.

In operating for carcinoma the general plan of resection is the same but an effort is made to remove as much of the omenta and related lymphatic field as possible. The greater omentum therefore should be divided close to the transverse colon, or, preferably, its inferior layer should be incised with the scalpel where it meets the colon, and the

entire omentum, the upper leaf of the transverse mesocolon and the peritoneum of the posterior wall of the lesser sac should be swept upwards off the back wall of the abdomen. The latter manœuvre is the decollement or "de-gluing" of the omentum of French surgeons. When the right part of the omentum is being freed up to the stomach it is important to include all the subpyloric glands in it and not to leave any in front of the pancreas. Troublesome bleeding may be encountered from division of small veins in this region and careful hæmostasis is required. The line of division of the lesser omentum should be as close to the liver as possible and sometimes it may be feasible to dissect glands off the hepatic artery and common bile duct as they proceed to the porta hepatis The left gastric artery must be taken at its origin from the cœliac in carcinoma cases and the lesser omentum stripped down off the upper part of the lesser curve and œsophagus, taking glands with it. Ideally the glands around the cœliac axis should also be removed by dissection and this is often possible at any rate in thin subjects. In dividing the stomach itself a margin of apparently normal gastric wall at least two inches proximal to the upper edge of the growth should be obtained Distally the resection should include at least one inch of duodenum beyond the growth.

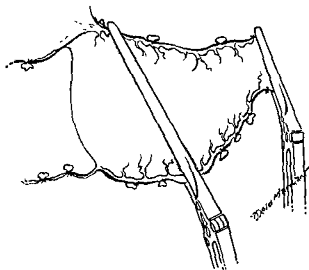


Fig 37 Billroth I gastrectomy Application of large Payr clamp across body of stomach at upper limit of resection

THE BILLROTH I OPERATION

The approach and initial stages of the operation are as for the Billroth II resection. The duodenum is mobilized and divided between clamps The duodenal stump is left controlled with its clamp and covered with a pack whilst the freeing of the stomach and preparation of the gastric stump are completed. The technique previously described is used up to the stage of division of the stomach. As the lesions for which the Billroth I operation is performed often lie high on the lesser curve, it is usually better to dispense with a proximal occlusion clamp on the gastric stump whilst the latter is being closed and anastomosed to the duodenum. Instead a large Payr crushing clamp is placed across the stomach from the lesser curve proximal to the ulcer or growth and usually three-quarters to one inch below the cardia, to the greater curve at a convenient point (*see* Fig. 37). With a gastric ulcer this may be well down in the body of the stomach, making the direction of the clamp very oblique indeed. A spring clamp is placed distal to the crushing clamp and the lesion and the stomach divided between the clamps with a scalpel which cuts flush with the Payr clamp. Starting at a point one-and-a-half to two inches from the greater curve and extending to the lesser curve a running mattress suture of No. 1/0 catgut is inserted with a straight needle just proximal to the Payr clamp (*see* Fig. 38). The unsutured lower third or quarter of the stomach cross-section is now controlled by another small Payr clamp applied obliquely as in Fig. 39, and the large clamp removed. A small wedge of stomach at the greater curve, between the two

clamps is excised. The upper sutured two-thirds of the stomach has only been closed by a mattress suture so far which has no hæmostatic effect. To ensure complete control of bleeding the catgut suture is returned as a closely placed over-and-over stitch along the edge till the small Payr clamp is reached, the insertion of this suture being facilitated by drawing on the latter to make the cut edge of the stomach taut. Finally the edge is buried by a series of interrupted Lembert sutures of fine silk (*see* Fig. 39).

In dealing with particularly high ulcers on the lesser curve, the surgeon may find it impossible to apply a large Payr clamp across the stomach proximal to the lesion. Two

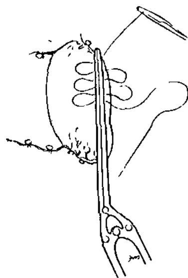


FIG 38 Billroth I gastrectomy Insertion of running mattress suture of catgut under Payr clamp

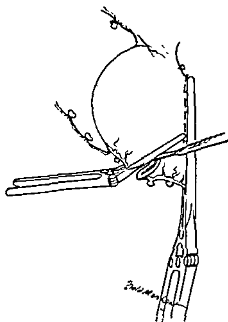


FIG 39 Billroth I gastrectomy. Application of small Payr clamp to lower part of stomach cross-section and excision of a wedge of redundant stomach adjacent to greater curve

alternative techniques are then available to him. He may place the clamp distal to the lesion and leave the latter in the retained gastric stump as recommended by Madlener (1939), or he may temporarily leave the ulcer in the stomach stump but before doing the anastomosis excise it by a step cut (Pauchet 1931). With the ideal operating conditions now provided by general anæsthesia combined with relaxant drugs, the former manœuvre should seldom be required, practically invariably it is possible to include the ulcer site in the resection.

The surgeon is now left with a duodenal and a gastric stump both of which are closed at their ends by crushing clamps. These clamps are approximated and the stumps anastomosed in conventional manner A light spring clamp is applied to each of the stumps, well away from the Payr clamp, and a posterior row of interrupted Lembert stitches of fine silk are inserted. It will be found easier to place these if they are all inserted loosely with the two stumps some distance apart before any of them are tied (*see* Fig. 41). The crushing clamps are then shaved off as in Fig. 42, leaving two open stumps, the cut edges of which are anastomosed by a continuous catgut stitch, first the posterior

halves and then the anterior halves. If a Connell stitch is used for the anterior through-and-through stitch it is important to catch and tie the individual vessels in the anterior edge of the stomach stump. The occlusive clamps are now removed and the anterior row of Lembert stitches is inserted.

In operating for gastric ulcers which have penetrated into the pancreas the surgeon may separate the stomach from the ulcer crater leaving the latter adherent to the pancreas,

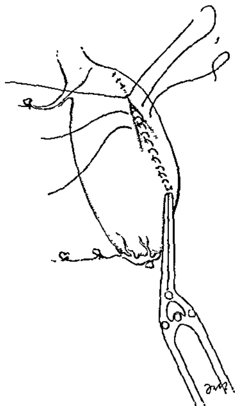


Fig 40 Billroth I gastrectomy After removal of the large Payr clamp and in-

tained on the small Payr clamp

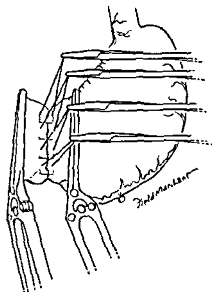


Fig 41. Billroth I gastrectomy. Apposition of duodenal and gastric stumps and insertion of posterior row of Lembert sutures of mattress type.

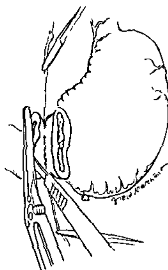


Fig 42. Billroth I gastrectomy. The controlling Payr clamps, together with the crushed tissues between their blades, are cut off by the scalpel leaving two open ends for approximation by a continuous catgut suture

and creating a corresponding hole in the part of the stomach stump which will be resected. No direct treatment of the ulcer crater is required; excluded from the alimentary tract it gives rise to no further trouble. If the surgeon is uncertain however as to whether the ulcer he is dealing with is benign or malignant it is essential to excise it completely; with a deeply penetrating lesion this will entail the performance of a formal pancreatic resection with splenectomy and total gastrectomy, which greatly increases the hazards of the operation. The same problem is encountered with a high lesser curve lesion in which malignancy cannot be included. If benign it can always be adequately excised by a partial gastrectomy with if necessary removal of some of the medial œsophageal wall,

but if malignant a total gastrectomy with spleno-pancreatic resection would be preferable. However the prognosis with gastric carcinoma even when radically treated is so poor that it scarcely seems justifiable to inflict on the patient with a probably benign lesion the greater operative risks of this more major operation. Each case obviously requires to be judged on its merits but in dealing with older subjects it will often be advisable to be content with the less drastic operation even though this involves a slight risk of treating a carcinoma inadequately.

BILLROTH GASTRECTOMY WITH JEJUNAL OR COLONIC REPLACEMENT. In order to provide a substitute for the normal gastric reservoir after gastrectomy and thereby minimize some of the physiological ill-effects of the operation, it has recently been suggested that continuity between the gastric and duodenal stumps should be re-established by interposition of a short segment of jejunum (Henly 1953) or transverse colon (Moroney 1953). In the present author's experience these methods have not yielded significantly better practised results and in ulcer cases have been frequently followed by the development of further ulceration in the transplanted portion of intestine, so that they cannot be recommended.

Abdominal Total Gastrectomy

This operation was introduced by Schlatter of Zürich in 1897, but till recent years was seldom employed because of the technical difficulties and formidable mortality associated with it. With modern anæsthetic and surgical technique it has been found to be an eminently practicable procedure.

Adequate access is usually provided by a left or right paramedian or median incision with the patient flat, and rarely is it necessary to add a transverse cut across one or both recti. A transverse curved or angled incision with the convexity upwards is a useful alternative in patients with a wide epigastrium. For cases in which there is doubt before exploration as to whether the resection can be completed solely through the abdomen, it is a good plan to have the patient on his right side tilted at an angle of 70 or 80 degrees to the horizontal and to commence the operation with a small transverse incision over the left rectus. This allows a hand to be introduced into the abdomen and a decision to be made regarding the operability of the lesion and the chances of resecting it entirely through the abdomen. If opening of the left chest is considered essential the incision is enlarged upwards along the ninth rib or interspace as a formal abdomino-thoracic approach. For the subsequent steps in the operation then *see* page 101, para. 3.

If on the other hand it is elected to perform an abdominal total gastrectomy the patient is turned into a supine position and the incision continued across the epigastrium. The initial part of the resection follows the lines of the Hofmeister-Finsterer partial gastrectomy for cancer with mobilization of the distal two-thirds of the stomach and division and closure of the duodenum. Attention is now turned to the upper part of the stomach. The gastro-splenic ligament is not divided; instead the peritoneum is severed with scissors behind the spleen and this organ mobilized forwards together with the tail and body of the pancreas and the splenic vessels. This process is continued almost to the junction of the splenic and superior mesenteric vein, the inferior mesenteric vein *being sacrificed. The splenic vein and artery are then ligated and divided and the body* of the pancreas cut across. The cervical end of the latter is sutured with non-absorbable material to control bleeding and diminish the escape of pancreatic juice from it. The

caudal part of the pancreas and spleen are now free except for their attachment by the gastro-splenic ligament and vasa brevia to the stomach and growth. A few strands of connective tissue and peritoneum at the top of the fundus require to be divided to complete the mobilization of the entire greater curve up to the lower end of the œsophagus.

On the lesser curve side the left gastric vessels have still to be taken. They should be divided as close to the cœliac artery as possible and their œsophageal branches severed on the medial side of the œsophagus and stripped down. Any obviously enlarged glands around the cœliac axis should be dissected away provided this can be done without jeopardizing the hepatic artery. The lower end of the œsophagus has now been completely bared on all aspects. Before proceeding to anastomose it to the intestine the anterior and posterior vagal trunks should be divided, for this allows the œsophagus to be drawn down for a further 1½–2 in. and permits of a higher level of proximal section.

The œsophagus may now be divided between clamps or may be retained temporarily intact to allow of the stomach being used as a means of traction till the posterior row of Lembert sutures between it and the intestine has been inserted. A variety of techniques exists for completing the operation. The œsophagus is usually anastomosed to the upper jejunum, the jejunal loop being brought up behind the transverse colon through the mesocolon. The different forms the actual anastomosis may take are sufficiently indicated by Fig 43 (*a*), (*b*), (*c*), (*d*). Alternatively a direct end-to-end œsophago-duodenostomy may be attempted and is more often feasible without tension than might be imagined. An isolated loop of jejunum (Henly 1952) or transverse colon (Goligher and Riley 1952) may also be used to bridge the gap between the œsophagus and the first part of the duodenum, it being hoped thereby to give the patient a substitute gastric reservoir and lessen the post-operative functional disturbances after total gastrectomy. In the performance of any of these anastomoses two rows of sutures, an inner of continuous fine No. 1/0 or 2/0 chromic catgut and an outer of interrupted fine silk or linen stitches, are satisfactory. In addition the jejunum or other piece of intestine used should be sutured to the peritoneum or diaphragm immediately behind and in front of the anastomosis to support the bowel and take the strain off the suture line. A Ryle's tube should be threaded through from œsophagus to intestine during the anastomosis, and retained for several days, as a means of avoiding jejunal distension by continuous or intermittent suction

Abdomino-Thoracic Total Gastrectomy and Proximal Partial Gastrectomy

See Volume II

Complications After Partial and Total Gastrectomy

The patient after gastrectomy is liable to all the complications of a major abdominal operation, and particularly to pulmonary collapse and infection; but he is exposed to certain additional hazards peculiar to the nature of the operation:

(1) **Peritonitis Due to Leakage from the Duodenal Stump.** This is most liable to occur in duodenal ulcer cases where it has been difficult to close the stump satisfactorily, but it occasionally follows gastrectomy for gastric ulcer or cancer where there has been no obvious technical difficulty with the stump. If a drain has been left down to the

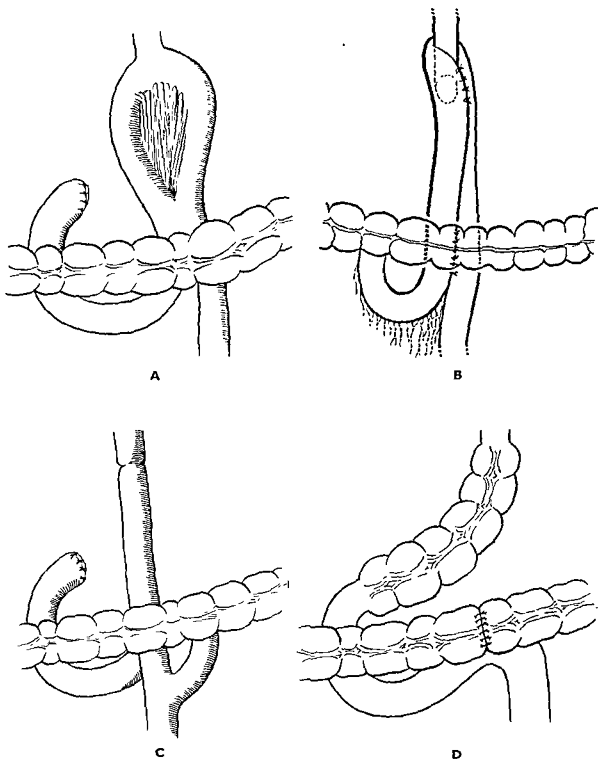


FIG 43 Diagrams showing four common methods of establishing continuity of the alimentary tract after total gastrectomy

(*a*) By end-to-side œsophago-jejunostomy and jejuno-jejunostomy
(*b*) By Roscoe Graham's modification of (*a*), sandwiching the lower end of the œsophagus between the two limbs of jejunum loop
(*c*) By end-to-end œsophago-jejunostomy and jejuno-jejunostomy (Roux's method)
(*d*) By interposition of middle thirs of transversed colon between œsophagus and duodenum

duodenum, the leakage proclaims itself by the escape of bile from the wound along the drain track, usually about the fifth to tenth day. When no drain has been employed the rupture usually causes sudden severe abdominal pain rather like that attending perforation of a duodenal ulcer, and the abdomen is found to be tender and guarded. Occasionally peritonitis develops more insidiously.

In drained cases where bile is already escaping from an external duodenal fistula no further operative treatment is required and the management is along the lines indicated on page 153.

If the abdomen was not drained at the time of operation, the development of signs of duodenal stump leakage or peritonitis will demand laparotomy. The opening in the end of the duodenum can sometimes be closed by further suture, but often the tissues are too friable to allow of this, and in any event the leakage usually recurs two or three days later. A drain should therefore be placed down to the stump and the conservative regime detailed above instituted.

(2) **Peritonitis Due to Leakage from the Anastomosis.** This is exceptionally rare after partial gastrectomy competently performed, but is unfortunately not at all infrequent from the œsophago-jejunal or œsophago-duodenal anastomosis after even the most skilfully executed total gastrectomy. Reliance has to be placed on spontaneous recovery after drainage, which should always be instituted at the time of the initial operation in total gastrectomy cases. The writer has had several patients who have survived after this form of leakage.

(3) **Post-Operative Gastric Retention or Vomiting.** A certain amount of gastric retention is not uncommon for the first two or three days after partial gastrectomy. It may be due to œdema at the stoma or may be part of a paralytic ileus. Usually it is controlled by gastric aspirations through a Ryle's tube as part of the routine post-operative treatment. If the plasma proteins are deficient this may contribute to stomal œdema and may be combated by plasma infusions. If retention should continue for several days the possibility of it being due to kinking or twisting of the efferent jejunal loop or some other obstructive mechanism must be considered, and laparotomy may be required.

(4) **Post-Operative Hæmorrhage.** Some degree of bloodstaining of the gastric aspirate is common for 24–36 hours after gastrectomy, but serious bleeding such as used to occur occasionally after gastro-enterostomy is very rare. 'If really severe hæmorrhage should take place it would require transfusion and possibly a further laparotomy

(5) **Pseudo-membranous Entero-colitis.** (*See* Chapter XIV)

The Physiological and Symptomatic Effects of Gastrectomy

Resection of a large part or all of the stomach produces certain physiological effects and symptoms, the severity of which is apparently roughly proportional to the amount of stomach removed.

Effects on Gastric Motor Function. After any form of gastrectomy sacrificing the pyloric sphincter, food passes rapidly into the small intestine causing sudden distension. This jejunal distension is believed to be responsible for the post-cibal sensation of epigastric fullness experienced by many gastrectomy patients in the early weeks after operation and known as the *dumping syndrome*. Sometimes a feeling of nausea, and faintness and sweating accompany the fullness. Usually the symptoms pass off in a few minutes or half-an-hour, but sometimes the patient has to lie down for a time. The

majority of patients after high partial gastrectomy and all patients after total gastrectomy suffer from some dumping symptoms for a few weeks, but these can be minimized by reducing the size of meals taken. Gradually the patients' capacity for food increases and after six or twelve months only a small minority of partial gastrectomy patients—perhaps 5–10 per cent—continue to have any real discomfort. Most of these consider that their dumping symptoms are not as inconvenient as the ulcer symptoms for which the operation was performed (Goligher and Riley, 1952). It is a moot point whether dumping occurs with equal frequency after the Billroth I and II technique of partial gastrectomy; the consensus of opinion is that it is probably somewhat less frequent after the Billroth I operation.

Another troublesome symptom which sometimes occurs after partial or total gastrectomy is *regurgitation of bile*. This may take place in association with dumping or may occur quite apart from it, perhaps at night or before or after meals. Usually only a mouthful of bile is brought up but sometimes the patients vomit as much as half a pint of pure bile immediately after taking food. Biliary vomiting only rarely occurs after the Billroth I operation and the vomitus then seldom amounts to more than half an ounce.

Effects on Gastric Secretions. The estimation of the acidity of the gastric contents after partial gastrectomy is confused by the difficulty of collecting satisfactory specimens unmixed with bile from the small active gastric remnant. This accounts for the variations in the results reported by different writers. Complete and permanent anacidity is apparently very common after partial gastrectomy for gastric ulcer; after high gastrectomy for duodenal ulcer it is said by Gaviser (1948) to occur in 85 per cent of the patients. Patients with stomal ulcers following gastro-enterostomy who are treated by subtotal gastrectomy generally show a continued hypersecretion of free hydrochloric acid after the resection.

Effects on Nutrition. Many patients after partial gastrectomy find difficulty in regaining their pre-operative weight. Total gastrectomy patients are much worse off in this respect; they tend to lose further weight in the post-operative period and usually become severely emaciated at any rate for a time. This nutritional disturbance appears to be due partly to the difficulty which these patients often experience in taking adequate meals on account of dumping symptoms, and partly to the impairment of fat absorption, which has been conclusively demonstrated by Wollaeger and his associates (1946) and by Brain (1951). Relatively large losses of fat may occur in the stools and this steatorrhœa may give rise to troublesome diarrhœa which further aggravates the patients' nutritional difficulties. Strangely enough digestion and absorption of protein and carbohydrate are usually not hindered by gastrectomy. Indeed glucose is often rapidly absorbed, causing an alimentary hyperglycæmia which is succeeded some two or three hours after the ingestion of the glucose by a hypoglycæmic phase which may cause constitutional and abdominal symptoms—*late dumping*.

Failure of absorption of Vitamin B may result in signs of deficiency after partial gastrectomy and much more commonly after total gastrectomy. These take the form of a glossitis or pharyngitis and sometimes after total gastrectomy the discomfort which they cause may produce severe dysphagia.

Effects on Blood Formation. It might be expected that after total gastrectomy the loss of the intrinsic anti-anæmic factor would inevitably result in the development of a

pernicious type of anæmia. As a matter of fact this is an extremely rare occurrence, perhaps because few of the patients undergoing a total gastric resection for cancer survive for any long period. Macrocytosis in the peripheral blood is not uncommon however. A mild secondary anæmia is occasionally encountered after partial gastrectomy or more commonly after total gastrectomy, and this usually responds to simple iron therapy.

Management of Postgastrectomy Syndromes. The important thing to bear in mind is the great tendency to spontaneous improvement. In severe cases small frequent meals with little accompanying fluid should be advised so that the total intake of food per day may be maintained at an adequate level. If necessary the patient may have to lie down for a few minutes after taking food. If steatorrhœa and diarrhœa are troublesome, the fat may be better absorbed if spread over all the feeds rather than concentrated in one or two main meals. For the few patients who continue to have severe dumping or bile regurgitation six to twelve months after operation the question of further operation is sometimes raised. Conversion of a Polya gastrectomy to a Billroth I type of anastomosis if technically feasible will certainly stop bilious vomiting and will probably minimize dumping symptoms but, as Perman (*see* Sjögren, 1952) has shown, it exposes the patient to a greater risk of recurrent ulceration. The establishment of an entero-anastomosis between the limb of the jejunal loop may also minimize bile regurgitation after Polya gastrectomy but is even more likely to predispose to stomal ulceration. Butler and Capper (1951) have recommended hitching up the lesser curve end of the gastric stump to the region of the left gastric artery or diaphragm as a means of eliminating dumping symptoms, but the value of this step has not yet been clearly established.

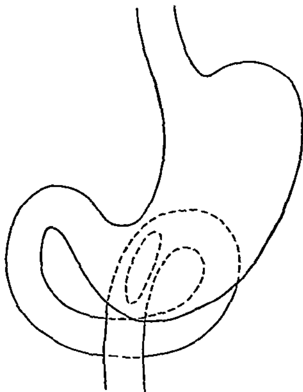


Fig. 44. Diagram of posterior gastro-enterostomy. Completed operation

Gastrojejunostomy

Gastro-enterostomy was originally performed for an obstructing carcinoma of the pylorus by Wölfler in 1881. He made the anastomosis between the anterior surface of the stomach and a long upper loop of jejunum, carried up in front of the transverse colon and mesocolon. This anterior or antecolic operation is still occasionally required as a palliative procedure for an inoperable growth, but, to avoid the kinking of the jejunal loop which was found to be a not uncommon complication causing troublesome vomiting, an entero-anastomosis, as recommended by Braun (1892), is now normally performed between the two limbs of the loop. Whilst it is safe to make this additional lateral anastomosis in a case with cancer and achlorhydria, in a patient with a duodenal

ulcer it is liable to be followed by jejunal ulceration due to the drainage of bile and pancreatic juice away from the anastomotic area. For the treatment of peptic ulcer which soon became the main indication for the operation the posterior no-loop gastro-enterostomy was developed by von Hacker, Petersen, Mayo and Moynihan (1926) (Fig. 44). The shortness of the jejunal "loop" minimized the risk of kinking and the necessity for entero-anastomosis with its undesirable consequences (*see* Fig. 44). The practice of narrowing the pylorus by sutures, or of transecting it as advocated by von Eiselsberg

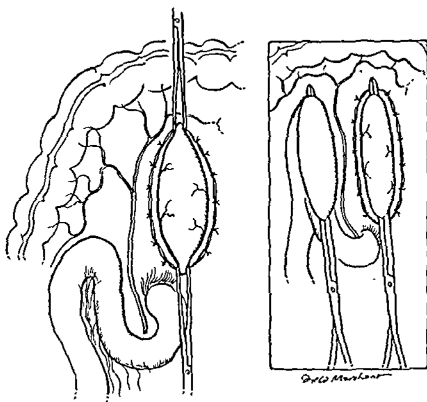


Fig 45 Posterior gastro-enterostomy An opening has been made in the transverse mesocolon to the left of the middle colic vessels and the posterior wall of the stomach has been drawn through and sutured to the edges of the gap (The upper loop of jejunum and the posterior surface of the stomach have been brought together controlled by spring clamps preparatory to anastomosis)

(1910), in association with gastro-enterostomy was soon given up as it was found to increase the incidence of stomal ulceration. The operation is now usually performed as follows:

The upper abdomen is explored and the existence of a duodenal ulcer established. The transverse colon and mescolon are lifted upwards and a vertical incision 3 in. long made in the mesocolon to the left or right of the main middle colic vessels. This exposes the posterior gastric wall which is made to protrude by the left hand invaginating the lower part of the front of the stomach. If it is desired to make the stoma in the stomach vertical—and this is the most favoured direction—two pairs of tissue forceps are applied to the posterior surface, one at the lesser curve near the incisura angularis and the other at the greater curve at its most dependent part (*see* Fig. 45). If an oblique or transverse

opening is preferred this can be accomplished by grasping the posterior gastric wall in an appropriate axis by the tissue forceps. These forceps are now raised so as to lift up a fold of stomach wall protruding through the rent in the mesocolon. The cut edges of the mesocolon are next sutured to the stomach all round. A spring clamp is then applied to the projecting portion of stomach (*see* Fig. 45). The duodenal-jejunal junction is sought and the first 8 or 10 in. of jejunum are milked empty and a spring clamp is now applied to this uppermost loop of jejunum, the proximal end of the clamp lying about 2 in from the duodena-jejunal flexure. The clamps holding the stomach and jejunum respectively are now approximated and packs placed around them.

A lateral anastomosis is established between the stomach and the jejunum using four rows of sutures. Firstly the stomach and bowel are approximated by a posterior row of interrupted fine silk or No. 2/0 chromic catgut sutures of Lembert type. Then, the jejunal wall is incised with cutting diathermy for 2½–3 in., the lumen being opened throughout that length. The stomach wall is next incised for a corresponding distance but in the first instance only the seromuscular layers are divided. The gastric mucosa with vessels ramifying on its outer surface now bulges outwards. The vessels along the anterior edge of the elliptical area can be underrun with fine catgut sutures on a small curved needle, or they may be caught with artery forceps so that when the mucosa itself is next incised the vessels may be tied off individually. The cut edges of the jejunal and gastric openings are now sutured by a running through-and-through stitch of No. 2/0 chromic catgut, which takes up the adjacent posterior edges first of all and then is continued round the front to approximate the anterior edges. In order to secure good inversion of the latter the anterior through-and-through stitch may be of the "loop on the mucosa" or Connell type. This stitch however has a poor hæmostatic effect and if it is used it is essential to take the precaution already mentioned of ligating individually the vessels on the gastric mucosa along the anterior edge of the stoma The posterior edges are of course well controlled by the over-and-over stitch used for the posterior through-and-through suture, and the jejunal wall does not usually bleed sufficiently to require special precautions. The spring clamps and surrounding packs can now be removed and the anastomosis is completed by insertion of an anterior row of Lembert sutures of silk or catgut. Lastly the edges of the rent in the transverse mesocolon are sutured to the posterior surface of the stomach half an inch or so from the actual anastomosis to prevent herniation of a loop of small bowel into the lesser sac.

Complications and Sequelæ of Gastro-Enterostomy

Hæmorrhage

Hæmorrhage in the immediate post-operative period may be taking place from the original ulcer for which the operation was performed, or more commonly it occurs from the cut edges of the stomach in the anastomosis. A slight amount of bleeding causing bloodstaining of the gastric contents aspirated from the stomach by the Ryle's tube is quite common during the first 24 or 36 hours after operation, but on occasions the bleeding may be profuse and the patient may have gross hæmatemeisi and melæna. Hæmorrhage from the suture line can be prevented by careful hæmostasis, as emphasized in the description of the technique of gastro-enterostomy. If hæmorrhage does occur but is mild it will probably soon cease and needs no special treatment. More severe bleeding will

require transfusion, and, if it should continue or assume alarming proportions, it may be necessary to re-open the abdomen, to incise the anterior wall of the stomach, and expose the anastomosis from within. The bleeding area on the suture line can then be underrun or ligated.

Hæmorrhage occurring months or years after gastro-enterostomy *usually comes from a stomal ulcer* and may be an important sign of the latter, but may also be due to an unhealed duodenal ulcer, a fresh gastric ulcer or a jejuno-gastric intussusception. It is treated along the lines already laid down for the management of gastroduodenal hæmorrhage in general.

Vicious Circle Vomiting

With the posterior no-loop operation this is a rare complication, but sometimes if the loop is made rather longer than usual it may sag down and become distended, with kinking of the efferent limb at the stoma. As a consequence the duodenal and jejunal contents find it easier to return to the stomach and so a vicious circle is established with resulting vomiting and dehydration. Sometimes however the mechanism is that the stoma becomes obstructed by œdema or the jejunum may be compressed by a loaded transverse colon. A mild degree of vomiting may respond to aspiration of gastric contents and intravenous infusions, but if the regurgitation continues for two or three days or is very severe the abdomen will have to be re-opened and the condition rectified. The classical procedure recommended for this complication is an entero-anastomosis between the limb of the jejunal loop but this is open to the serious objection that it leaves the anastomosis exposed to undiluted gastric juice. Unless the patient's condition is desperate therefore it is better to undo the gastro-enterostomy and establish another with a shorter jejunal loop or better sited stoma, or to do a partial gastrectomy.

Peritonitis

Peritonitis after gastro-enterostomy may be due to leakage from the suture line—an excessively uncommon occurrence after efficient surgery—or more probably to perforation of the duodenal ulcer for which the operation was performed or of a jejunal ulcer developing subsequently. Perforation calls for laparotomy and suture.

Strangulation of a Loop of Small Bowel in the Window in the Mesocolon

If the opening in the transverse mesocolon is not securely sutured to the posterior wall of stomach all round the anastomosis a loop of jejunum or ileum may be herniated through it into the lesser sac and become obstructed and strangulated. This complication is prevented by care at the original operation and if it occurs is treated like any other acute intestinal obstruction.

Jejuno-Gastric Intussusception

Occasionally the loop of jejunum anastomosed to the stomach is intussuscepted in retrograde fashion through the stoma into the stomach and becomes obstructed. Either both limbs of the loop or one or other of them is affected and the complication can occur after the posterior no-loop operation or after anterior gastro-enterostomy;

it has also been recorded in cases where an entero-anastomosis had been performed. Usually it occurs some months after the operation and causes the symptoms of an acute upper intestinal obstruction with copious vomiting, sometimes bloodstained. Operation is soon called for and the precise nature of the obstruction is usually in doubt till the abdomen has been opened. Usually the intussusception does not undergo strangulation.

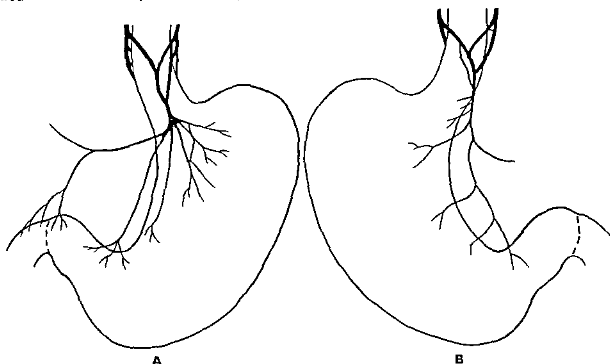


FIG 46 Anatomy of vagi in relation to abdominal vagotomy·
(*a*) Anterior vagal trunk (after McCrea) (*b*) Posterior vagal trunk (after McCrea).

There is also a more chronic or recurrent form with remission of symptoms, which are usually dismissed as due to a badly functioning stoma, till careful radiological examination during an attack shows the intussusception.

JEJUNAL OR STOMAL ULCER

(*See* page 103)

Vagotomy

Vagotomy is never used as a sole procedure now except in patients suffering from stomal ulceration following an adequate partial gastrectomy, and even here the surgeon usually wishes to open the abdomen to verify the diagnosis. Vagotomy is therefore practically always performed by the abdominal route (*see* Fig 46 (*a*), (*b*)) and the original transthoracic vagotomy is now obsolete. Only under one circumstance may the latter method still be required and that is when the presence of impenetrable adhesions resulting from numerous previous operations renders abdominal vagotomy impossible. Even then, however, by using an extraperitoneal approach to the hiatus between the diaphragm and diaphragmatic pleura it is usually quite possible to achieve a satisfactory vagotomy by the abdominal route. If the surgeon is not disposed to adopt this technique the vagal plexus on the thoracic œsophagus can be exposed and resected for 1 in. or so through a formal postero-lateral thoracotomy removing the right or left eighth rib.

Abdominal Transhiatal Vagotomy. A high left paramedian incision gives good access. The diagnosis of duodenal or stomal ulcer is confirmed. The hiatal region is then exposed by retracting the left lobe of the liver strongly upwards with a broad, deep retractor. This gives as satisfactory an approach to this region as does the more commonly employed method of dividing the left triangular ligament and turning the left lobe aside under the right. A transverse cut is made with scissors through the anterior peritoneum just below the hiatus and the œsophagus is exposed. By blunt dissection with the finger it is freed so that a rubber tube may be passed round it and used for traction (*see* Fig. 47 (*a*)).

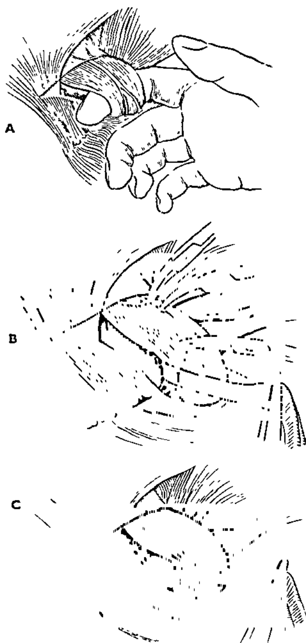


FIG. 47. Vagotomy by the abdominal, transhiatal route
(*a*) Division of peritoneum and hooking down of œsophagus
(*b*) Resection of anterior vagal trunk
(*c*) Exposure of posterior vagal trunk by retraction of œsophagus

The œsophagus is now pulled down firmly and the anterior vagal trunk sought. Usually it is easily felt as a taut cord on the front of the œsophagus an inch or so above the cardia, but sometimes it seems to lie more to the right. Lower down it splits into a right and a left branch, and if it should divide abnormally early two trunks will be found instead of one anterior vagus. Two long artery forceps are now applied to the main trunk of the anterior vagus, about 1 in. apart (Fig. 47 (*b*)), and a piece of nerve resected from between them for histological examination. The forceps are then touched with the diathermy electrode to coagulate the ends and seal any contained vessels and axon sheaths.

The posterior vagal trunk has next to be identified. To find it the œsophagus is drawn forwards and to the left and the nerve sought first of all in the œsophageal bed, rather to the lesser curve side, and if not discovered there, on the back of the œsophagus itself (Fig. 47 (*c*)) The posterior vagus is a stouter nerve than the anterior vagus and remains a single trunk for a greater distance. It is divided in exactly the same manner as that used for the anterior nerve.

Finally, a careful search is made for any other abnormal vagal fibres that may be present, especially in front of the œsophagus. When the surgeon is satisfied

that all filaments have been severed the rubber tube is withdrawn. The peritoneal cut may be sutured if desired but this is not necessary.

If the vagotomy is being used in support of some other operation such as a gastro-enterostomy this is now carried out and the abdomen is then closed

Complications After Vagotomy. The complications of vagotomy used in conjunction with gastro-enterostomy or gastrectomy, or for stomal ulcer following these operations, are essentially those of the associated procedure.

Simple Suture of Perforated Peptic Ulcer

The upper abdomen is entered by a right paramedian or median incision, the diagnosis usually being instantly confirmed by the escape of gas or bile-stained fluid from the

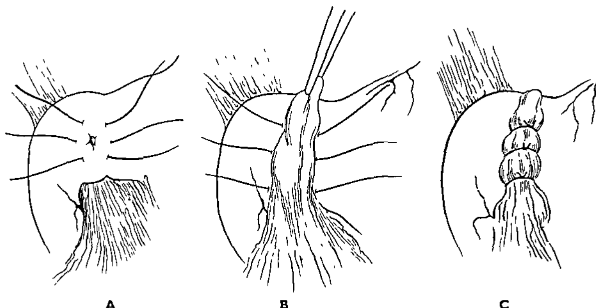


FIG 48 *(a), (b), (c)* *Closure of perforation by Roscoe Graham's method, incorporating a tongue of greater omentum in the through-and-through sutures*

peritoneal cavity. The perforation is then sought. Usually it will be found on the anterior wall of the duodenum or pylorus, and the first step consists of exposing this adequately by having the assistant retract the right edge of the wound and draw the stomach to the left. If no perforation is present there, the lesser curve is carefully examined, and if necessary the lesser sac opened by dividing the greater omentum so that the posterior wall of the stomach and duodenum may be searched. The perforation may be closed in a variety of ways—by a purse string suture; by three or four interrupted Lembert sutures inserted vertically or horizontally; by three or four through-and-through sutures passed wide of the actual perforation in a horizontal axis and tied so as to include a tongue of greater omentum (Roscoe Graham's method *see* Fig. 48); by plugging the perforation, if very large, with a piece of greater omentum, or lastly, if all other methods fail, by suturing a large rubber tube into the stomach as a temporary gastrostomy which can be used for suction or feeding, the resulting temporary gastric fistula usually closing spontaneously. The closure of the perforation by several of these methods can be reinforced by suturing the greater and lesser omenta over it.

The fluid in the peritoneal cavity is removed as far as possible by suction from the pelvis, subhepatic and subphrenic spaces, penicillin-sulphonamide powder is applied to the wound and peritoneum, and the abdomen closed in layers usually without peritoneal drainage but sometimes with a drain to the parietal wound.

Ramstedt's Pyloro-Myotomy

The child comes to the theatre with its limbs wrapped in cotton wool and bound to a T-shaped metal splint, the arms being in an abducted position and the abdomen exposed. In the majority of cases local infiltration anæsthesia down to the peritoneum in the line of the proposed incision suffices, the child being at the same time comforted by an occasional sip of glucose water. Rarely is it necessary to supplement this with light nitrous oxide and oxygen anæsthesia.

The abdomen is opened by a high median or right paramedian incision overlying the pyloric tumour, which is then brought to the surface with a finger or with the help of special forceps designed by Denis Browne. An incision is now made through the peritoneum on the front of the tumour and into the superficial part of the muscle. The division of the muscle down to the mucosa is completed by stretching with forceps rather than by cutting for fear of perforating the latter. The danger of opening into the lumen is greatest at the distal end where the "duodenal fornix" may easily be opened in dividing the muscle. It is important therefore to be especially careful in this region and not to carry the incision farther distally than the pyloro-duodenal vein. When the myotomy has been completed the mucosa should bulge freely through the elliptical muscular defect. The stomach is now squeezed gently to see if air can be made to pass freely into the duodenum and also to demonstrate that no mucosal perforation has been produced. Any breach of the mucosa is immediately repaired with a few interrupted catgut sutures and usually gives rise to no trouble. The pylorus is returned to the abdomen and the wound closed in layers.

Pyloroplasty

This operation has recently been revived to some extent in conjunction with vagotomy. There are several ways of performing it but the simplest and perhaps the best is that associated with the names of Heineke and Mikulicz (1888). In this technique a longitudinal incision is made through the anterior wall of the narrowed pylorus and first part of the duodenum for a distance of $1\frac{1}{2}$–2 in and opens into the lumen. Each edge of this wound is seized by a tissue forcep about its middle and retracted strongly converting the longitudinal opening into a lozenge shaped one which is then sutured in this transverse direction, the suture line being tied with a series of Lembert sutures.

Gastrostomy

As this operation is not required for lesions of the stomach but for the relief of œsophageal obstruction it is dealt with in a later volume

BIBLIOGRAPHY

Allison, P. R. and Borrie, J. (1949) *Brit. J Surg.* 37, 1.
Balfour, D. C. and McCann, J. C (1930) *Surg. Gynec. & Obstet.* 50, 948.
Bancroft, F. W. (1932) *Amer J Surg* 16, 223.
Barrett, M. K. (1946) *J. Nat Cancer Inst* 7, 127.

Beattie, J. (1932) *J. Canad. Med. Ass.* **26**, 278.
Berkson, J., Walters, W., Gray, H. K., and Priestley, J. T. (1952) *Proc. Staff Meetings Mayo Clinic*, **27**, 138.
Billroth, T. (1881) *Wien. Med. Wschr.* **31**, 161.
[illegible]
Braithwaite, L. R. (1923) *Brit. J. Surg.* **11**, 7.
Braun, H. (1892) *Arch f. klin. Chir.* **45**, 361.
Brunschwig, A. (1951) *Surg. Gynec. & Obstet* **92**, 375.
Butler, T. J. and Capper, W. M (1951) *Brit. med. J.* **1**, 1177.
Carman, R. D. (1913) *J. Amer. med Ass.* **61**, 321.
Castle, W. B. (1929) *Amer. J. Med. Sci.* **178**, 746.
Castleman, B. (1936) *Ann. Surg.* **103**, 348.
Code, C. F. and Varco, R L. (1940) *Proc. Soc. Exp. Biol. and Med.* **44**, 475.
Coller, F. M., Kay, E. B., and McIntyre, R. S. (1941). *Arch. Surg.* **43**, 743.
Cooper, W. A. (1949) *Surgery*, **23**, 425.
Cushing, H. (1932) *Surg. Gynec. & Obstet* **55**, 1.
Davies, D. T. and Wilson, A. T. M. (1937) *Lancet*, **2**, 1353.
Devine, H. B. (1928) *Surg. Gynec. & Obstet.* **47**, 239.
Dible, J. H. (1924) *Brit. J. Surg.* **12**, 666.
Doll, R. (1950) *Brit med J.* **1**, 215
Doll, R. and Jones, F. Avery (1951) *Med. Research Council.* Special Report No. 276 Occupational [illegible] s. *H.M. Stat. Off.*, London.
[illegible] 73, 676.
Dragstedt, L. R., Woodward, E. R., Harper, P. V. Jr., and Storer, E H. (1948) *Gastroenterology*, **10**, 200
Eiselsberg, von A. (1910) *Wien klin Wschr.* **23**, 44
Eker, R. (1951) *Acta Chir. Scandin.* **101**, 112
Eusterman, G. B and Sentry, E. G. (1922) *Surg Gynec & Obstet.* **34**, 5.
Farmer, D. A., Howe, C. W., Porell, W. J , and Smithwick, R. H. (1951) *Ann Surg.* **134**, 319.
Fibiger, J (1913) *Ztschr. f. Krebsforsch* **13**, 217
Finsterer, H (1914) *Deutsche Ztschr. Chir* **128**, 514
Finsterer, H. (1918) *Zentralblatt. f Chir.* **45**, 434
Finsterer, H (1936) *Lancet*, **2**, 303.
Finney [illegible]
C [illegible]
C [illegible]
C [illegible]
Goligher, J. C. and Riley, T R (1952) *Lancet*, **1**, 630.
Gordon-Taylor, G (1935) *Lancet*, **2**, 811.
Graham, R. R (1940) *Surgery*, **8**, 257
Grossman, M. I. (1951) in Peptic Ulcer, p 165 Edited by D J. Saundweiss. *W. B. Saunders and Co.*, Philadelphia and London.
Haberer, von H. (1919) *Wien klin. Wschr.* **32**, 413.
Haberer, von H (1920) *Arch klin. Chir.* **114**, 127.
Habe [illegible] . 80, 195.
Habe [illegible] 72, 496
Hack [illegible] 616.
Harn [illegible]
Henl [illegible]
Heslop [illegible]
Hoerr, [illegible]
Hofme [illegible] 351
Hosford, J. (1949) *Brit. med J.* **1**, 929.
Hurst, A. F. (1911) The Sensibility of the Al [illegible]

Ivy, A. C. (1920) *J. Amer. med. Ass.* **75**, 1540.
Ivy, A. C. and Fauley, G. B. (1931) *Amer. J. Surg.* **11**, 531.
James, A. H. and Pickering, G. W. (1949) *Clin. Science*, **8**, 181.
Jamieson, J. K. and Dobson, J. F. (1907) *Lancet*, **1**, 1061.
Johnson, H. D., (1950) in Techniques in British Surgery. Edited by Rodney Maingot. *W. B. Saunders and Co.*, Philadelphia and London.
Johnson, H. D. and Orr, I. M. (1953) *Lancet*, **1**, 253.
Jones, F. Avery (1947) *Brit. med. J.* **2**, 441 and 477.
Jordan, S. (1951) in Peptic Ulcer. p. 539. Edited by D. J. Saundwiss. *W. B. Saunders and Co.*, Philadelphia and London.
Kaplan, H. S. and Rigler, L. G. (1945) *Amer. J. Med. Sci.* **209**, 339.
Klein, A. J. and Palmer, W. L. (1941) *J. Nat. Cancer Inst.* **1**, 559
Kronlein, R. V. (1888) *Corr. Schw. Aerzte*, **18**, 317.
Lake, N. C. (1937) *Brit. med. J.* **2**, 49.
Latarjet, A. (1922) *Bull. Acad. de méd. Paris*, **87**, 681.
Levin, E., Kirsner, J. B., Palmer, W. L., and Butler, C. (1948) *Gastroenterology*, **10**, 952.
Longmire, W. F. Jr. (1947) *Surg. Gynec. & Obstet.* **84**, 21.
Lowdon, A. G. R. (1948) *Edin. med. J.* **55**, 533.
Madlener, M. (1939) *Zentralblatt. f. Chir.* **66**, 360.
Maingot, R. H. (1948) *Ann. Roy. Coll. Surg.* **3**, 248
Maingot, R. H. (1953) Management of Abdominal Operations. *H. K. Lewis*, London.
Mann, F. C. (1951) in Peptic Ulcer p. 103. Edited by D. J. Saundweiss. *W. B. Saunders and Co.*, Philadelphia and London.
Mann, F. C. and Williamson, C. S. (1923) *Ann. Surg.* **77**, 409.
McIver, M. A. (1927) *Ann. Surg.* **85**, 704
McKittrick, L. S. and Moore, F. D. (1945) *Ann. Surg.* **120**, 531.
Mikulicz, J. (1888) *Arch. f. klin. Chir.* **37**, 79.
Moroney, J. (1953) *Ann. Roy. Coll. Surg. Eng.* **12**, 328
Moynihan, B. (1926) Abdominal Operations. 4th Ed., *W. B. Saunders and Co.*, Philadelphia.
Newcomb, W. D. (1933) *Brit. J. Surg.* **20**, 279
Nissen, R. (1945) Duodenal and Jejunal Peptic Ulcer. *Wm. Heinemann*, London.
Ogilvie, W. H. (1947) *Lancet*, **2**, 377.
Palmer, W. L. (1926) *Arch. Int. Med.* **38**, 694.
Pannett, C. A. (1938) *Surg. Gynec. & Obstet.* **67**, 485
42, 424
is.
Péan, J. (1879) *Gaz. d'hôp.* **52**, 473
Pfeiffer, D. B., (1941) *Surg. Gynec. & Obstet.* **72**, 282.
Plenk, A. (1936) *Zentralblatt f. Chir.* **63**, 3019.
Polya, E. (1911) *Zentralblatt. f. Chir.* **38**, 892
Polya, E. (1940) *Surg. Gynec. & Obstet.* **70**, 270
Pyrah, L. N. (1935) *Proc. R. Soc. Med.* **27**, 233
Rammstedt, C. (1912) *Med. Klin.* **8**, 1702
Reeves, T. B. (1920) *Surg. Gynec. & Obstet.* **30**, 374
40, 1
Schindler, R. (1937) Gastroscopy. *University of Chicago Press*, Chicago.
Shoemaker, J. (1911) *Arch. f. klin. Chir.* **94**, 541
Sjögren, O. (1952) *Nord. med.* **47**, 747
Stewart, M. J. (1931) *Lancet*, **2**, 565
St. John, F. B., Swenson, P. C., and Harvey, H. D. (1944) *Ann. Surg.* **119**, 225.
Strong, L. C. (1945) *J. Nat. Cancer Inst.* **5**, 339
Swynnerton, B. F., and Truelove, S. C. (1952) *Brit. med. J.* **1**, 287.
Tanner, N. C. (1950) *Proc. R. Soc. Med.* **43**, 147
Tanner, N. C. (1951) *Brit. med. J.* **2**, 47.
Taylor, H. (1941) *Lancet*, **2**, 276
Taylor, H. (1951) *Proc. R. Soc. Med.* **44**, 279
Thompson, J. (1921) *Edin. med. J.* **26**, 1.

Troisier, E. (1889) *Arch. gen. med.* 1, 122.
Visick, H. (1948) *Lancet*, 1, 505 and 551.
Walton, A. J. (1930) Surgical Dyspepsia. 2nd Ed. *Edward Arnold*, London.
[illegible]
Willis, R. A. (1953) Pathology of Tumours. 2nd Ed., *Butterworth & Co., Ltd.*, London.
Wilson, L. B., and McCarty, W. M. (1909) *Am. J. Med. Sc.* 138, 846.
Wolf, S., and Wolff, H. G. (1943) Human Gastric Function. A Study of Man and his Stomach. *Oxford University Press*, New York and Toronto
Wolfler, A. (1881) Über die von Herrn Professor Billroth ausgefuhrten Resectionen des Carcinomatosen Pylorus. *W. Braumuller*, Wien.
Wolfler, A. (1881) *Zentralblatt. f. Chir.* 8, 705.
Wollaeger, E. E., Comfort, M. W., Weir, J. F., & Osterberg, A. E. (1946) *Gastroenterology*, 6, 93.
Wright, G. (1935) *Brit. J. Surg.* 22, 433.
Yudin, S. (1939) *J. Internat. de Chir.* 4, 219
Zinninger, M. M. and Collins, W. T. (1949) *Ann. Surg.* 130, 557.

CHAPTER III

SECTION I

THE LIVER AND BILIARY TREE

J. M. PULLAN

INJURIES OF THE LIVER

THE liver is normally protected by the ribs and is not often injured by direct trauma. When the abdomen is penetrated, however, it is frequently involved as a result of its large size. The injury may be closed or open.

Closed Injuries

These are caused by the sudden application of force over a wide area of the body surface perhaps fracturing the protecting ribs. The force may be a blow, a crush, or blast. The liver substance may be disrupted beneath an intact capsule resulting in a subcapsular hæmatoma, later rupturing and leading to intra-abdominal hæmorrhage or biliary peritonitis. More extensive injury causes long splits in the liver substance or even complete detachment of large portions of the organ. The damage may underlie the site of the violence or may be caused by contre-coup. The liver may become displaced through a simultaneously injured diaphragm.

Open Injuries

These are more commonly seen in time of war when the abdomen is penetrated or traversed by metallic fragments or bullets. Civilian knife wounds account for some of the cases in less peaceful localities. According to the weapon the liver wound may be clean and precise or ragged, contaminated and disruptive. Missiles traversing the porta hepatis may sever the hepatic arteries, bile ducts or portal vein resulting in necrosis of part of the organ in those who survive the immediate hæmorrhage. These wounds are often abdomino-thoracic

Treatment. Wounds of the liver are not in themselves so important as the direct results of the wound—hæmorrhage, biliary leakage and the possibilities of associated damage to other viscera.

The probability of a liver wound can often be deduced from the direction of the missile track; or the seeping of bile to the surface may suggest the severance of a major bile channel. The remaining signs are those of hæmorrhage and peritonitis which are not specific to liver wounds The diagnosis of a closed wound is more difficult and may be found only at laparotomy Even when the presence of a liver wound is certain it is very difficult to exclude injury to other viscera and the signs of peritonism resulting from even mild hæmorrhage may be quite indistinguishable from a true early peritonitis due to laceration of the duodenum. It is for this reason that laparotomy is often indicated apart from the actual evidence of hepatic damage.

If the liver alone is injured there is no immediate need for laparotomy provided that blood loss may be balanced by transfusion. If the wound is not gross, clotting soon ensues, which with the pressure of the abdominal tone will arrest the hæmorrhage.

If the blood loss cannot be compensated, or if, after repeated and very careful observations, the signs suggest the onset of early peritonitis (as judged by increasing rigidity, tenderness and abdominal silence) laparotomy should be performed through an upper right paramedian incision. Where there is also evidence of penetration of the thoracic cavity and there are indications for opening this, a left or right thoraco-abdominal incision may be required.

The abdomen is carefully searched for injury to other viscera and in their absence attention confined to the liver. Hæmorrhage may increase as a result of opening the abdomen and if severe may require immediate packing while transfusion is accelerated. Meanwhile both portal vein and hepatic artery are compressed in the free border of the lesser omentum. When the patient has rallied or hæmorrhage is less severe the extent of the liver damage is determined. Loose or partially severed parts of the liver are removed as there is a danger of uncontrolled infection (particularly that due to Clostridium Welchii) in avascular liver tissue.

Hæmorrhage from remaining lacerations is controlled by means of long mattress stitches parallel to the raw surface and placed in close apposition. The capsule itself may be sutured where it can be approximated without tension. If there is a tendency for the mattress sutures to cut through, omentum or detached muscle fragments may be inserted between the catgut loops and the capsule (*see* section on operative surgery). Oxycel gauze, though probably increasing the risk of sepsis, may be included in the raw surfaces as an adjuvant to hæmostasis where this cannot otherwise be secured. If all else fails the liver wound may be packed with gauze around which the abdominal wound is closed. This gauze is removed with some care two or three days later when it is hoped that the hæmorrhage will not be repeated. These wounds should in any event be drained as leakage of bile is almost inevitable.

Foreign bodies are removed only if they come to hand These patients will not usually withstand exhaustive attempts to locate and reach fragments in inaccessible parts of the body. Their removal is frequently unnecessary, or they can be dealt with later should their presence be causing prolonged sepsis or a sinus. These operations should be covered by full dosage of antibiotics and anti-gas-gangrene serum may have a place.

Post-operative complications include peritonitis (biliary or primary pyogenic), subphrenic or hepatic abscess, biliary fistula and secondary hæmorrhage.

References

(1) *Brit. J. Surg.* War Supplement No. 3.

LIVER ABSCESS

SUPPURATION within the liver occurs in a variety of circumstances and in response to different agencies. For descriptive purposes these entities are often divided into watertight compartments; but to the clinician the diagnosis is first liver abscess, and the elucidation of its ætiology is a secondary, though important, consideration. For this

reason the subject matter is here treated as for liver abscess as a whole and not as for a series of unrelated diseases.

Ætiology. The liver is vulnerable to infection through a number of different routes and by a variety of organisms. These may reach the liver in the following ways.

(i) Portal Vein

Usually there is already a focus of infection in the portal territory, such as an acute appendicitis, diverticulitis, ulcerative colitis, dysentery, peptic ulcer or suppurating hæmorrhoids. Organisms gain access to the terminal portal venous branches and are carried to the liver where they are, for the most part, destroyed or eliminated in the bile. A few may gain a hold, however, and suppuration develops. Although liver abscesses may occasionally be caused in this way, more commonly there is an actual suppurative thrombophlebitis in the terminal venules from which infected emboli separate and lodge in the liver. This is portal pyæmia. Sometimes the thrombotic process may extend for some distance along the portal vein—portal pylephlebitis.

In the same way liver abscess may rarely follow relatively clean abdominal operations such as gastrectomy, closure of colostomy, resection and anastomosis of intestine. In such cases the bacterial flora reflects the source of the infection and a mixture of representative intestinal organisms is usually found. b.coli, streptococcus fæcalis, staphylococci and B. Welchii are typical of this type of abscess which is commonly multiple.

The same route is used in amœbic migration from the colon to the liver.

Actinomycosis and hydatid disease almost certainly reach the liver via the portal vein.

(ii) Umbilical Vein

This is probably the avenue of infection of the liver in infants secondary to umbilical sepsis and thrombophlebitis. The liver abscess may be multiple or single, with or without septicæmia.

(iii) The Bile Ducts

Cholangitis is a common accompaniment of acute or chronic cholecystitis, especially when there is a common duct stone. The condition becomes more serious in the presence of biliary obstruction, whatever the cause, as the back pressure and stagnation render the hepatic tissue vulnerable to bacterial attack The organisms found in these cases are again of an intestinal pattern, though b.coli is the usual culprit. The cholangitis characteristically fluctuates but at the height of any one attack actual suppuration may begin within the liver substance. This is normally a widespread, punctuate process and abscesses are consequently disseminated throughout a bile stained liver.

It was thought that organisms ascended the common bile duct from the duodenum, and while this possibility is not excluded, it is more likely that they reach the ducts from the liver. Even the normal liver passes live organisms into the bile whence they are returned harmlessly to the intestine. In the presence of stagnation or stone these organisms cease to be harmless and cause inflammation of the bile ducts, canaliculi and liver parenchyma.

(iv) The Hepatic Artery

When there exists a systemic septicæmia or pyæmia, the liver, in common with other organs, shares the risk of abscess formation Staphylococcus aureus or the hæmolytic

streptococcus are the organisms concerned in most cases. The abscesses are often multiple, and pale into insignificance or may be unrecognized amidst the dramatic and widespread manifestations of the disease elsewhere.

There exists, however, another type of abscess which is solitary and arises without any septicæmic signs or symptoms. The organism is present in pure growth and is often of low virulence, such as the streptococcus viridans.

In such cases it seems unlikely that organisms gained access by a route other than the hepatic arteries, though where they came from often remains unknown. The very rare tuberculous and typhoid abscesses of the liver probably reach it in this way.

(v) Direct Spread

Suppuration may spread into the liver from contiguous inflammations. The gall bladder is the most frequent source, though this is rare. A subphrenic abscess seems to burrow into the liver on occasions, though the converse is the more likely direction of spread.

(vi) Lymphatics

There is no reason why organisms should not reach the liver via the lymphatics, though it is hard to prove that this has occurred and such a hypothesis remains largely theoretical

(vii) Penetrating Injuries

Infected material may be carried into the liver by violence from outside the body. In war wounds and knife injuries such an event is not uncommon and intrahepatic suppuration may ensue.

Closed injuries may also initiate such a process for by contusion the liver substance may become inflamed by the organisms already within it.

Suppuration may also arise in pre-existing abnormalities within the liver, such as secondary carcinoma, hæmangioma, and cyst.

Classification. Liver abscesses may therefore be classified as follows:

(1) Venous

A. Portal	(*a*) Pyogenic	(i) secondary to abdominal sepsis.
		(ii) following abdominal surgery.
	(*b*) Amœbic.	
	(*c*) Actinomycotic.	
	(*d*) Hydatid.	
B Umbilical.	Secondary to omphalitis.	

(2) Cholangitic	Secondary to infection of the biliary tract.
(3) Systemic.	(*a*) Septicæmic and pyæmic.
	(*b*) Bacteræmic from distant focus.
	(*c*) Source unknown.
(4) Traumatic.	(*a*) Penetrating injuries.
	(*b*) Closed injuries.
(5) Miscellaneous.	Suppuration in pre-existing liver abnormalities.

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This is probably the avenue of infection of the liver in infants secondary to umbilical sepsis and thrombophlebitis. The liver abscess may be multiple or single, with or without septicæmia.

(iii) THE BILE DUCTS

Cholangitis is a common accompaniment of acute or chronic cholecystitis, especially when there is a common duct stone. The condition becomes more serious in the presence of biliary obstruction, whatever the cause, as the back pressure and stagnation render the hepatic tissue vulnerable to bacterial attack. The organisms found in these cases are again of an intestinal pattern, though b.coli is the usual culprit. The cholangitis characteristically fluctuates but at the height of any one attack actual suppuration may begin within the liver substance. This is normally a widespread, punctuate process and abscesses are consequently disseminated throughout a bile stained liver.

It was thought that organisms ascended the common bile duct from the duodenum, and while this possibility is not excluded, it is more likely that they reach the ducts from the liver. Even the normal liver passes live organisms into the bile whence they are returned harmlessly to the intestine In the presence of stagnation or stone these organisms cease to be harmless and cause inflammation of the bile ducts, canaliculi and liver parenchyma

(iv) THE HEPATIC ARTERY

When there exists a systemic septicæmia or pyæmia, the liver, in common with other organs, shares the risk of abscess formation. Staphylococcus aureus or the hæmolytic

PLATE I

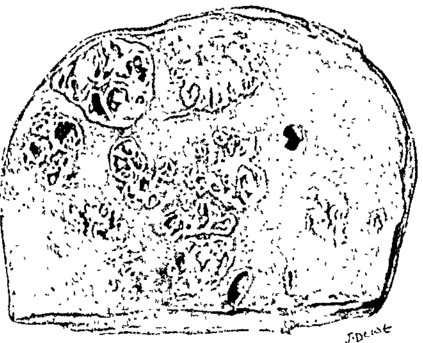


FIG. 50 Cross section of liver showing suppurative cholangitis

Morbid Anatomy. Abscesses are usually multiple and small in cases resulting from portal pyæmia (Fig. 49), suppurative cholangitis (Fig. 50) and septicæmia, and are disseminated throughout the liver. A solitary abscess is more likely to be amœbic, pyæmic, actinomycotic, or idiopathic, though no rules can be laid down. As the small abscesses enlarge and coalesce relatively large cavities are formed. These may reach the capsule causing a localized peritonitis and obliteration of the peritoneal space. In the same way the diaphragm may be penetrated and pleura involved. Sometimes this results in empyema or a lung abscess with a bronchial fistula. Large abscesses may rupture into the pleural, peritoneal or pericardial cavities or into any of the neighbouring viscera, or may point on the skin and there discharge.

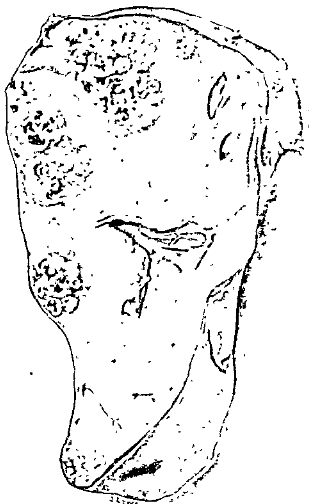


FIG 49 Cross section of liver showing multifocal coalescent abscesses from a case of portal pyæmia

The right lobe is the more usual site of single abscesses in the proportion of about 4 to 1, and is probably explained by its greater bulk, without invoking any theories with regard to the streaming effects of the portal blood flow.

Amœbic abscesses are more often single and show a predilection for the upper right zone, though they may be found anywhere in the liver (Fig. 3). They usually start as minute foci of liquefaction in a liver already the seat of amœbic hepatitis which enlarge and later coalesce giving one or more large abscesses. The pus is characteristically like anchovy sauce, especially in acute cases, and is composed of blood, lysed liver cells and few leucocytes, differing from true pus in this way. It is normally sterile, and the Entamœbæ histolytica exist almost entirely in the wall of the abscess which eventually may become encapsulated and even calcified in very long standing cases. Secondary infection may occur and it then assumes more of the characteristics of a pyogenic abscess.

Actinomycotic abscesses also form by the coalescence of smaller areas, and the disease is often confined to one quadrant of the liver (Fig. 51). The abscess is honeycombed and seldom cavitates completely. The pus contains the "sulphur granules" which are the colonies of the actinomyces bovis. These abscesses tend to be less acute than the pyogenic ones and, respecting no anatomical barriers, burrow throughout the tissues reaching the

PLATE I

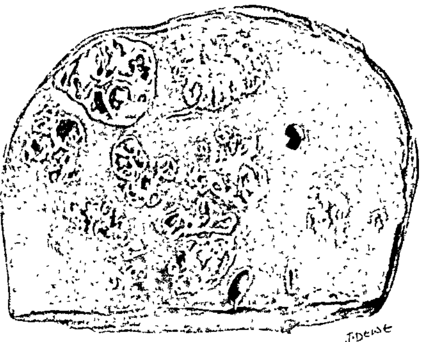


FIG 50 Cross section of liver showing suppurative cholangitis.

surface or neighbouring skeletal tissues such as muscle and even bone. The liver infection is nearly always secondary to a focus elsewhere in the portal field, and the commonest site is the ileo-cæcal region and appendix.

Clinical Features. Whatever the cause of the infection there are certain features common to all abscesses of the liver.

Mostly they arise during the course of another disease and their features are thus added to symptoms and signs already present. The onset may on this account be relatively insidious, but usually the patient is more ill than can be explained on the basis of the original pathological condition. The expected improvement is not achieved and the patient's condition steadily deteriorates. He is usually haggard with a muddy moist facies and perhaps later mildly icteric. Emaciation is rapid and accompanied by anorexia and vomiting. Both pulse and temperature show a gradual swinging rise often punctuated by rigors and with profuse sweating. There is usually upper abdominal aching with sometimes a sharp pleuritic-like pain when peritoneum is involved, which is referred to the flank, abdomen or shoulder. Some patients complain of a persistent boring pain over the lower thoracic spine.



FIG 51 Large ragged amœbic abscess of liver.

There is tenderness over the liver, which progressively enlarges both upwards and downwards so that the diaphragm is seen to be elevated and less mobile on screening, and the liver edge is found below its proper level in the abdomen thus enlarging the area of liver dullness. The surface may be palpably irregular or fluctuant and a peritoneal rub is occasionally heard. Mild icterus may be present and the urine contain bile pigments, though this is not necessarily so.

The abscess may not remain confined to the liver and sometimes finds the subphrenic spaces and even penetrates the diaphragm and pleura, causing empyema if the pleural space is not already sealed, or alternatively tracks into the lung securing a form of drainage into the bronchial tree when the pus is expectorated. An amœbic abscess is most prone to this last resort.

Subcutaneous œdema may overly the abscess which eventually points on the surface. There is a marked leucocytosis with a predominance of granulocytes; secondary anæmia is common.

There is a more silent type of abscess usually arising without any obvious source of infection which may smoulder undetected for many weeks, producing only generalized symptoms. The organism may be relatively avirulent or have been attenuated by chemotherapy or antibiotics during either blind therapy or the deliberate treatment of a previous malady. The signs are slight, but in time the evening pyrexia, cachexia, pain and a swollen tender liver will lead to the diagnosis, particularly when the abscess reaches the liver capsule and local signs become more obvious. These abscesses are unlikely to be multiple.

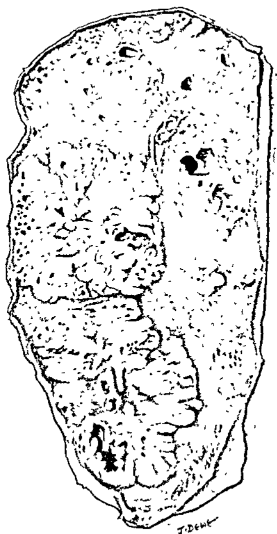


Fig. 52 Actinomycosis the honeycomb arrangement and widespread burrowing disease are evident

An amœbic abscess may not differ clinically from a pyogenic abscess, and the two are often confused up to the time of examining the pus. There are those which start with an acute hepatitis with an enlarged, tender liver, abdominal rigidity, pain and marked pyrexia. Prostration and vomiting is severe, and this state may pass imperceptibly to abscess formation which can sometimes be recognized by localized deformation of the liver. There is a leucocytosis in acute cases with granulocytes usually under 80 per cent. Alternatively the abscess may develop very quietly and reach considerable proportions years after leaving the Tropics. Such patients are anæmic and cachectic and cannot eat. They may have no symptoms directing attention to the liver. There may be no leucocytosis

Diagnosis. When an abscess is diagnosed it is hard to know whether it is intrahepatic or subphrenic though such a distinction is not vital as one may lead to the other. The subphrenic is the more susceptible to treatment and carries a better prognosis. Where there is doubt the more optimistic view should be taken in the first instance. Antecedent abdominal sepsis may lead to either, though a frank perforation is far more likely to cause a subphrenic abscess which in any case is more common than one in the liver. There may be no certain way to diagnosis other than by exploratory operation.

Amœbic and pyogenic abscesses are usually described as belonging to two watertight compartments. However true this may be in pathology it is seldom so clinically and the first task, since the treatment is different, is to exclude amœbiasis as a cause.

It may be that there are already clear pointers to the source of infection. If there has

been a recent appendicitis or other intra-abdominal sepsis the liver infection is almost certainly due to a portal pyæmia or pylephlebitis. If, in the presence of obstructive jaundice or during the course of an acute cholecystitis, the symptoms and signs of hepatic suppuration appear a cholangitic origin is indicated. With longstanding disease in the right iliac fossa, perhaps with sinus formation, actinomycosis as a cause of the liver abscess must be considered. Nevertheless, it should not be forgotten that cholecystitis, appendix abscess, ileocæcal actinomycosis or cæcal amœbiasis may all show similar signs in the right iliac fossa and yet each give rise to a different type of liver abscess.

When hepatic pain and fever come out of the blue without any preceding symptoms the abscess is likely to be systemic, though in many cases the portal of entry will remain unknown.

Where such histories are available there will be little difficulty in reaching a correct diagnosis. Unfortunately many patients are seen without such clues and in these careful inquiry should be made for tropical residence or previous dysentery. Amœbic abscess may occur up to twenty or more years following the original infection, which in any event will have passed unnoticed in at least half of those who develop an abscess. Nor is residence abroad essential as there are well authenticated cases arising in this country and there are probably many carriers. In the United States of America estimates of the carrier state have been given as high as 20 per cent of the population. The stools must be examined repeatedly on the warm stage for amœbæ containing red cells. Cysts may be seen in the quiescent phase containing not more than four nuclei.

Sigmoidoscopy should be carried out and a search made for ulcerated areas or other evidence of intestinal infestation Negative results to all of these investigations do not exclude a diagnosis of amœbiasis, and the most reliable test is the response to emetine hydrochloride which should be given in daily intramuscular doses of 1 gr. for five days. If the liver abscess is amœbic the response should be dramatic as judged by signs and the pyrexia, though a secondarily infected abscess may not show a complete response. All cases of liver abscess for which there is no satisfactory explanation should be subjected to this test.

Amœbic abscess formation arises in an area of hepatitis and it is often impossible to say just when a hepatitis becomes an abscess unless there is a demonstrable bulge on the liver surface. At times the presence of an abscess is suggested when the result of medical treatment falls short of a cure. If an amœbic abscess is secondarily infected it will be indistinguishable from a pyogenic abscess, which in fact it is, though the history or the stool examination may have revealed the underlying cause. Amœbic abscess is the most treatable and carries the best prognosis of all liver abscesses, especially if open drainage is avoided, and for this reason it is vital to exclude it before proceeding to more active treatment.

In non-endemic countries a hydatid basis for hepatic suppuration will not be suspected in the absence of a previous history of infestation until hydatid elements are recognized in the pus or the cyst wall is encountered during a drainage operation Hydatid disease of the liver is considered below.

Liver abscess may also be confused with the following:

(1) Acute cholecystitis.
(2) Virus hepatitis.
(3) Acute pyelitis.

(4) Secondary carcinoma.
(5) Other pyrexial states of undetermined ætiology.

Treatment

(i) Pyogenic Abscess

There are two effective methods of treatment, by antibiotics and chemotherapy, or by drainage, and some combination of the two may be required.

(*a*) Antibiotics and Chemotherapy. This method nearly always constitutes the first line of attack as most cases pass through a stage when the diagnosis is no more than probable but the urgency of symptoms demand some form of treatment. However bad in principle, such action is often no more than prudent. Furthermore, during the evolution of any liver abscess there is a stage during which the process can be aborted by energetic therapy and no time should be lost in applying it. In this way many cases are caught before the damage is too great, and frank suppuration requiring operation is thus avoided.

In other cases the diagnosis may be more certain—a full blown abscess is already present. Even here, though operation will be necessary, full dosage with antibiotics should be started immediately in the hopes of attenuating or containing the infection. It may well be that unexpected improvement will follow and operation become less urgent. While improvement continues drainage may be delayed until a standstill is reached. Such an expectant policy is justified by the high incidence of multiple abscesses in this category for which operation holds little promise owing to the physical impossibility of draining more than a few small cavities. In these cases chemotherapy is the only hope.

The organisms responsible are often mixed and many are gram negative and insensitive to Penicillin. Others rapidly acquire a resistance to the antibiotics used. More than one drug should therefore be given simultaneously. Full dosage of any two of the following should be employed—aureomycin, chloromycetin, penicillin, streptomycin or sulphadiazine. This treatment, if successful, should continue until at least ten days after the pulse and temperature have settled as there is a danger of a liver infection seeming to be cured only to break out again after the cessation of active antibacterial measures. Such a case requires drainage.

When pus is available following drainage a more certain choice of antibiotic can be made from the culture and bacterial sensitivity tests.

(*b*) Drainage. When one or more large cavities containing pus are present drainage will be required though the complexity of achieving it increases with the number of cavities Where cholangitis precedes abscess formation no time should be lost in establishing external T-tube biliary drainage by means of choledochostomy as there is almost always some degree of biliary obstruction present. Furthermore, the abscesses being small and multiple can only be drained in this way. Such patients are usually jaundiced prior to the formation of the abscesses. A cholecystectomy or cholecystostomy may also be required where an acute cholecystitis is the precipitating cause.

Localization. In order to plan a drainage operation it is advantageous to know where the abscess lies and this is not always easy. Some information can be obtained

where the abscess underlies the abdominal wall or lower ribs and is palpable or actually pointing or causing œdema.

An X-ray of the chest, both postero-anterior and lateral, may show a disproportionate diaphragmatic bulge into the thorax or displacement of abdominal viscera. There may be an associated subphrenic abscess with a fluid level, or air may enter those abscesses which communicate with a bronchus.

The site of pain may be of considerable assistance, particularly the sharp "pleuritic" type in the flank which often by its reference indicates that the abscess has reached the capsule and parietal peritoneum or pleura somewhere in that corresponding segmental territory. The more frequent incidence of right sided abscess is also a guide.

It may be tempting to tap the liver with an aspirating needle as an adjunct to localization This should be resisted unless it is done under direct vision at the time of operation, or just before operation. There is a real danger of peritonitis or empyema attendant upon such measures, although the liver can be reached posteriorly through the bare area without transgressing the serous cavities.

OPERATION. The drainage of a liver abscess is largely a question of approach. The available routes may be classified as follows:

Extra-serous

(1) Anatomical.	Through the bare area.
(2) Anatomical and pathological.	Subcostal with extraperitoneal tunnelling to site of adhesions. (*a*) Twelfth rib bed. (*b*) Kocher type incision.
(3) Pathological	Direct approach through area of pointing abscess.

Trans-serous

(1) Across pleura after preliminary pleurodesis—two stage
(2) Across peritoneum with omental seal—extraperitoneal tunnelling.

In draining a liver abscess it is desirable to achieve it without contaminating the pleural or general peritoneal cavities. This principle can be satisfied where the peritoneal cavity is locally obliterated by a subphrenic abscess or the adherence of the liver to the parietes, or drainage is effected through an area devoid of serosa. If a subphrenic abscess is known to be present it is approached in a manner appropriate to the location of that abscess, it is desirable at the same time to explore with the finger for a track extending from the subphrenic cavity into the liver and to enlarge this by stretching so that it will admit an adequate tube of about ¾ inch in diameter, which is then brought out through the wound of access.

When there is no certain evidence of a subphrenic abscess or adhesion of the liver to the parietes (as judged by the pointing of the abscess externally) the best approach is through an upper abdominal paramedian incision. At this time the liver is examined. It may be found that access to certain parts of the liver surface is denied by recent adhesions (Fig. 53 (*a*)), in which case the abdomen is closed and drainage effected by incising from the exterior through this obliterated area. Should the pleura be encountered during the making of this new approach then this cavity must be artificially sealed. This

is sometimes done by stitching parietal to diaphragmatic pleura in the region of the costo phrenic angle, but does not always provide an immediate seal that is absolutely safe (Fig. 53 (*b*)). A more certain method is to pack with iodine gauze the unopened parietal against the diaphragmatic pleura and discontinue the operation at this stage until in three or four

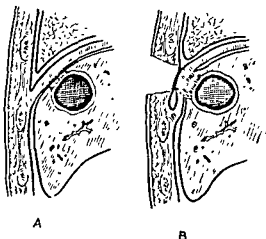


Fig 53 (*a*) An abscess of the liver dome has obliterated the subphrenic space where it has surfaced (*b*) Transpleural approach with preliminary pleural seal by sutures This can also be achieved by extrapleural packing with iodine swabs a few days before drainage—not recommended

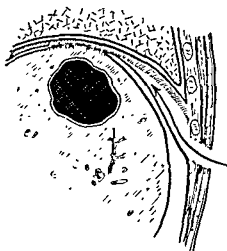


Fig. 54. Anterior extraperitoneal subphrenic approach to an abscess of the dome with local cœlomic obliteration

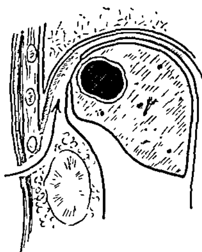


Fig 55. Posterior subcostal retroperitoneal approach to bare area or any high posterior abscess.

days' time adhesions have formed sufficient to allow safe drainage through the area without risk of empyema. This method involves some delay and is unsuitable where more urgent drainage is required. Another method and probably the best is to reach the abscess by tunnelling extraperitoneally from the abdominal incision between the diaphragm and parietal peritoneum to the area where the cœlom is obliterated by adhesions and hence through the adherent layers straight into the liver substance, inserting a drain along this

track into the abscess, which is tapped with the finger. This method is applicable to all cases and may be started from a Kocher or posterior (Fig. 54) subcostal incision.

Alternatively, at the initial laparotomy the liver surface may be everywhere accessible, but an abscess palpated beneath the surface. This can be verified by aspiration and the cavity emptied in this way. A finger is then thrust into the cavity, trabeculæ broken down and a large tube inserted. The greater omentum is brought up and the tube threaded through it and brought to the exterior at a point overlying the opening. During this procedure great care is taken to avoid contamination of the rest of the peritoneum and viscera by packing off the area.

In high placed anterior or lateral abscess the drain may be led extraperitoneally to below the costal margin as in the tunnelling method, crossing the cœlom directly and for a short distance only at the point of puncture of the abscess.

If an abscess is found either preoperatively or at laparotomy to be posteriorly situated it is best approached through the right twelfth rib bed with mobilization of the pleural reflection upwards (Fig. 55). The finger is directed upwards against the inferior diaphragmatic surface and the tissues anteriorly carefully felt for abnormal consistency. If an abscess is encountered the finger is pushed through the wall. This is sometimes called the approach through the bare area, though there is no means of locating this accurately.

Finally it may be found that the condition of the portal vein and the number of widespread liver abscesses defeat any attempt at drainage, in which case the abdomen must reluctantly be closed. Nevertheless it is always worth while to have verified that a drainable abscess was not being neglected.

In the presence of suppurative cholangitis and jaundice it is seldom possible preoperatively to say when abscess formation occurs. In the first instance it is essential to be sure of adequate external biliary drainage by a rapid method, such as cholecystostomy or choledochostomy. This should be combined with antibiotic treatment. In the majority of patients with suppurative cholangitis their condition will now improve. When, however, the expected improvement is not obtained, or after an initial remission deterioration sets in, liver abscesses are likely to be present, but are almost certain to be multiple, then the best hope is that they will drain down the biliary tract while the infection is fought by other means.

Management of the Tube. The principles are the same as for empyema. That is to say that the tube remains while there is yet any cavity to drain. After a week or ten days the tube is replaced by a thinner one of equal length which is passed down the lumen of the first. The larger tube is then withdrawn from around the thinner one, which should be about the width of a pencil. This is retained until the cavity closes around it and only a track remains. This can be judged by recourse to lipiodol and X-rays when the progress of the cavity closure can be observed. The tube may then be shortened daily until it is finally removed. The track rarely leaks any bile as the liver tissue is everywhere sealed by granulations and fibrosis.

(ii) Actinomycosis

There is not the same urgency for drainage in abscess due to this cause when the true nature of the infection is known. This disease has most frequently arisen as a portal

metastasis from intestinal actinomycosis which is usually in the ileocæcal region, and knowledge of the presence of such a lesion may lead to a correct appraisal of the liver pathology. Nevertheless a mass in the iliac fossa may equally be a pyogenic appendicitis and the liver involvement due to a portal pylephlebitis. The actinomycotic lesion is less acute and spreads more insidiously and less dramatically. As a result there is less danger of a rupture of the abscess but more tendency to a burrowing in all directions irrespective of anatomical barriers. The cavities are thus complex and, on account of the honeycombing, drainage at one point seldom drains the whole. Furthermore, antibiotics combined with sulphonamides and possibly radiotherapy hold out more hope for a cure than can be expected from surgery, which should be confined to the local and superficial attention to abscesses which point. On occasions there may be a secondary infection by b.coli, streptococci or staphylococci, and their presence adds to the seriousness of the infection and makes drainage more useful, though the secondary invaders are usually mild.

Many liver abscesses are drained in ignorance of their actinomycotic basis as the primary intestinal lesion may have disappeared. They may continue to discharge for many weeks before a search is made for the fungus or sulphur granules in the pus and their presence explains the chronicity of the lesion. A recent case followed operation for perforated gastric ulcer.

(iii) Amœbic Abscess

Whenever a liver abscess is diagnosed but the source of the infection is unknown the possibility of its being amœbic should be entertained and if such a diagnosis seems at all possible it should be subjected to proof by the exhibition of emetine. This is given in daily doses of 1 gr. of the hydrochloride subcutaneously. The test should be continued for at least five days; if there are unmistakable signs of improvement the evidence for an amœbic basis is very strong indeed and the course should be continued for a total of ten days and followed by a full medical regime.

It is vital to recognize an amœbic abscess before any drainage operation is carried out as the prognosis is adversely affected when drainage results in secondary infection. Open drainage is therefore contraindicated and the main reliance is placed upon the strong specific measures of emetine and chloroquine. This certainly applies to all cases of amœbic hepatitis, and those in whom there is early softening in the inflamed liver tissues, as many small abscesses are cured by emetine alone. This can be combined with antibiotics if secondary invaders are suspected. There will remain, however, certain abscesses so large that the volume of the contents is greater than can readily be absorbed, and also those in whom medical treatment has achieved only incomplete resolution. For these some form of emptying will be required after a period on emetine treatment.

Aspiration is the method of choice since it avoids secondary infection and the amœbic infestation of a wound. It is safer than in pyogenic abscess as secondary bacterial infection at the time of aspiration is uncommon and the results of a single aspiration are often curative. The manœuvre is carried out in the theatre under local or general anæsthesia with a long wide bore aspirating needle and Potain's syringe. The needle is directed to the suspected area and if the cavity is struck it should be emptied. Two or more aspirations may be required at intervals of a few days. The introduction of emetine into the cavity has been advocated

DRAINAGE. The pus obtained is subjected to immediate microscopy. If organisms are present and secondary infection is established immediate open drainage is indicated as for pyogenic abscess, using the needle as a guide. Amœbæ are difficult to find in the aspirated material. Secondary infection is suggested by a leucocytosis in which the granulocytes exceed 80 per cent of the total count.

Open drainage is also indicated:

(1) When medical treatment and repeated aspiration have failed and persistent deformity of the liver outline remains. It is estimated that there is more than one abscess in 45 per cent of cases and the presence of a second cavity may explain the failure.

(2) When an abscess exists in the left lobe and requires to be emptied. Aspiration is difficult and uncertain in such localities.

(3) Following intraperitoneal rupture which presents rather like a perforated ulcer. This can sometimes be anticipated.

Rupture into the pleura occurs but is said to respond to aspiration and drugs. More frequently the abscess burrows into the lung and drains into a bronchus. This is not an indication for operation. Open operation is seldom necessary today, the combination of emetine, chloroquine and aspiration prove effective in all but a very few and in these open drainage scarcely adds to the likelihood of cure when more effective measures have already failed. The bowel infection requires attention.

Prognosis

PYOGENIC ABSCESS

This will depend on the following factors:

(1) The danger from associated lesions elsewhere.
(2) Whether the abscess is single or multiple.
(3) Accessibility of the abscess without infecting pleura or peritoneum.
(4) Early diagnosis.
(5) Organisms present and their sensitivities.
(6) The presence of complications such as rupture.

The mortality varies enormously according to the underlying condition and death rates between 30 per cent and 90 per cent have been quoted.

AMŒBIC ABSCESS

The prognosis depends on:

(1) Efficient antiamœbic treatment.
(2) The general condition of the patient.
(3) The presence of alcoholism.
(4) Size of abscess.
(5) Accessibility of the abscess for aspiration.
(6) Spontaneous secondary infection.
(7) Secondary contamination of the draining wound.
(8) Number of abscesses.
(9) Virulence of the infestation.

The mortality depends upon the method required to combat the disease. Those responding to medical treatment, including aspiration, should not have a mortality of more than 5 per cent. When the case is severe enough to demand drainage the mortality may be expected to increase up to 50 per cent.

References

Pyogenic abscess:

(1) Eliason, E. L. (1926) *Surg. Gynec. Obstet.* 42, 510.
(2) Ochsner, A., De Bakey, M. and Murray, S. (1938) *Amer. J. Surg.* 40, 292.

Amœbic abscess.

(1) Brown, P W. and Hodgson, C. H. (1938) *Amer. J. Med. Sci.* 196, 305.
(2) Harinasuta, C. (1951) *Indian Med. Gaz.* 86, 137.
(3) Ochsner, A. and De Bakey, M. (1943) *Surgery*, 13, 460.
(4) Sodeman, W. A. and Lewis, B. O. (1945) *J. Amer. med. Ass.* 129, 99.

HYDATID DISEASE OF THE LIVER

Ætiology. This tape worm, Echinococcus granularis, exists predominantly in the small intestine of the dog, and although other animals may also carry it the dog is the important host so far as human medicine is concerned. It is only 5 mm. in length and consists of a head or scolex with four suckers and as many as fifty hooklets which enable it to fasten onto the intestinal mucosa of the host. The last segment contains ova which are shed into the intestinal lumen and passed in the fæces. They contaminate the ground and are eaten by sheep in which the hydatid cyst develops, which on the death of the sheep is consumed by other dogs—thus completing the life cycle.

Other animals may harbour both the worm and the hydatid and one of these is man who in this instance understudies the sheep and constitutes the alternative accidental intermediate host. This is a tangent from the main cycle as there are few opportunities for the human hydatid to return again to the dog. So far as man is concerned the dog is the source of infestation and the disease is most prevalent where man and dog exist in closest symbiosis. A large proportion of the British cases come from Wales, and Australian surgeons have the greatest opportunities of treating the disease. It seems that in childhood the danger of infestation is greatest when the fondling of dogs and sucking of fingers are prevalent. The ova hatch into small hexacanth (six hooked) embryos and penetrate the wall of the alimentary canal entering the portal vein radicles and are carried to the liver. Here they lodge in the hepatic capillaries and gradually form a unilocular primary hydatid cyst. A few may pass through the liver and lodge in the lung capillaries and those that pass through the lung are distributed systemically into any tissue in the body. Thus, 75 per cent of all hydatids are found in the liver, 15 per cent in the lung and the remainder throughout the rest of the body. More than one cyst exists in 60 per cent of affected patients.

Structure. The cyst wall has three layers—an *outer adventitial coat* which is contributed by the host and consists at first of compressed hepatic, pulmonary or other tissue, later becoming progressively thicker, more fibrous and perhaps even calcareous. The *middle coat* is the laminated membrane of the parasite. This consists of hyaline laminæ formed seriatim from the inside by the inner or germinal layer. There is always, in uncomplicated cysts, a cleavage plane between host and parasite and the outer surface of the laminated layer is shining and moist and faintly blue. The laminated membrane is under a certain stress not only from the pressure of its fluid content (up to 300 mm. of mercury) but also from its own structure, by which the outer and older layers are

PLATE II



FIG 56 Section of liver showing hydatid (hepatic) cyst.

References

Pyogenic abscess:

(1) Eliason, E. L. (1926) *Surg. Gynec. Obstet.* 42, 510.
(2) Ochsner, A., De Bakey, M. and Murray, S. (1938) *Amer. J. Surg.* 40, 292.

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HYDATID DISEASE OF THE LIVER

Ætiology. This tape worm, Echinococcus granularis, exists predominantly in the small intestine of the dog, and although other animals may also carry it the dog is the important host so far as human medicine is concerned. It is only 5 mm. in length and consists of a head or scolex with four suckers and as many as fifty hooklets which enable it to fasten onto the intestinal mucosa of the host. The last segment contains ova which are shed into the intestinal lumen and passed in the fæces. They contaminate the ground and are eaten by sheep in which the hydatid cyst develops, which on the death of the sheep is consumed by other dogs—thus completing the life cycle.

Other animals may harbour both the worm and the hydatid and one of these is man who in this instance understudies the sheep and constitutes the alternative accidental intermediate host. This is a tangent from the main cycle as there are few opportunities for the human hydatid to return again to the dog. So far as man is concerned the dog is the source of infestation and the disease is most prevalent where man and dog exist in closest symbiosis. A large proportion of the British cases come from Wales, and Australian surgeons have the greatest opportunities of treating the disease. It seems that in childhood the danger of infestation is greatest when the fondling of dogs and sucking of fingers are prevalent. The ova hatch into small hexacanth (six hooked) embryos and penetrate the wall of the alimentary canal entering the portal vein radicles and are carried to the liver. Here they lodge in the hepatic capillaries and gradually form a unilocular primary hydatid cyst. A few may pass through the liver and lodge in the lung capillaries and those that pass through the lung are distributed systemically into any tissue in the body. Thus, 75 per cent of all hydatids are found in the liver, 15 per cent in the lung and the remainder throughout the rest of the body. More than one cyst exists in 60 per cent of affected patients.

Structure. The cyst wall has three layers—an *outer adventitial coat* which is contributed by the host and consists at first of compressed hepatic, pulmonary or other tissue, later becoming progressively thicker, more fibrous and perhaps even calcareous. The *middle coat* is the laminated membrane of the parasite. This consists of hyaline laminæ formed seriatim from the inside by the inner or germinal layer. There is always, in uncomplicated cysts, a cleavage plane between host and parasite and the outer surface of the laminated layer is shining and moist and faintly blue. The laminated membrane is under a certain stress not only from the pressure of its fluid content (up to 300 mm. of mercury) but also from its own structure, by which the outer and older layers are



Fig 56 Section of liver showing hydatid (hepatic) cyst

more stretched by growth of the cyst than the more recent inner layers, so that the smallest puncture is liable to form the starting point of an extensive rent and the cyst will turn itself inside out. The rupture may then be almost explosive and no doubt these features serve a purpose in the dissemination of the live contents. The laminated layer is resistant to leucocytes, digestion and bacteria.

The *inner layer* is the germinal layer which secretes the laminated layer on the outside and the fluid within. It is about 1 cell thick and forms buds which protrude into the cavity. These become the brood capsules containing scolices and hooklets of potential worms and are just visible as "Hydatid sand" to the naked eye. They are not found in very young cysts.

The hydatid fluid is quite clear and contains little protein but is decidedly antigenic, producing severe anaphylaxis in sensitized subjects. It is used (from a sheep) for the Casoni intradermal test for hydatid sensitivity. It contains floating scolices and brood capsules which if liberated through rupture are each capable of forming a new univesicular cyst where they come to rest. For these reasons the fluid is highly dangerous and should be treated with the very greatest respect. These are *secondary cysts* and will be found distributed according to the manner of rupture of the primary cyst, whether intraperitoneal, intrapleural, intrapericardial, intravenous or intracardiac. Rupture into the intestine or bronchus does not seem to result in secondary cyst formation. It has been estimated that secondary cysts will form in some 20 per cent of those cases in which fluid is spilled at operation on pulmonary hydatids. These secondary cysts are external to the original primary cyst though they may be within the original adventitia in certain cases of local rupture

Daughter cysts may form within the original primary cyst. They may be regarded as internal secondary cysts and seem usually to arise owing to a failure or partial leakage of the laminated membrane which is common in older cysts.

RATE OF GROWTH. The disease is mostly contracted in childhood and the hydatid is as old as the man, or nearly so. The growth rate varies with the tissue in which it lies being greater in *more compressible structures of a yielding nature. The rate of growth* is checked by setbacks, such as leakage, which threatens the life of the parasite. Growth is very slow and at the end of the first year a cyst may attain a diameter of 2 cm

Hepatic Cysts

Like most metastatic hepatic lesions the right lobe is affected in the ratio of 4 to 1 as compared with the left. More than one cyst exists in the liver in 25 per cent of cases. The lower part of the liver contains three quarters of all hepatic cysts and these will in general be palpable. They may reach a large size and yet cause no symptoms. Seventy-five per cent of patients are obliged to seek advice because of complications in a previously silent cyst (Fig. 56, Plate II)

Complications. The older a cyst becomes the more certainty is there of complications. These are—

(I) RUPTURE

Leakage and especially rupture of a cyst calls forth an allergic reaction which may be severe or even fatal—urticaria, dyspnœa, asthma, syncope and vomiting may ensue.

(*a*) INTRAHEPATIC. This may be local and may pass with no more than minor allergic manifestations.

(*b*) INTRAPERITONEAL. This is a complete rupture with discharge of the contents into the peritoneal cavity. There may be severe shock and signs suggesting a perforated ulcer which quickly pass away. Biliary peritonitis may later set in. Secondary cysts eventually grow in the pelvis and throughout the cœlom.

(*c*) INTRABILIARY. The cyst bursts into a large duct of the biliary system. Daughter cysts may block the duct causing obstructive jaundice and mild biliary colic. This state may persist for some time as one cyst after another or hydatid debris is passed. The particles may be recognized in the stools as they are relatively indigestible. Suppurative cholangitis may follow.

(*d*) INTRAPLEURAL. This may lead to empyema and secondary echinococcosis of the pleural cavity. A bilious leak often accompanies the rupture giving a cholothorax. On occasions the pleural cavity is sealed and the rupture then takes place into a bronchus with expectoration of hydatid debris and bilious sputum; or air may enter the hepatic cavity giving a perivesicular pneumocyst. A lung abscess may be an intermediate stage.

(*e*) INTRAINTESTINAL. This is not uncommon.

(*f*) OTHER SITES. Rupture has been described into the pericardium and inferior vena cava.

(ii) SECONDARY INFECTION

Suppuration may arise around and in a cyst as a result of its earlier death, though the presence of pus or calcification cannot be taken as a guarantee that there are no live hydatid elements. Suppuration may precede the rupture into the peritoneum or pleura and these cavities are infected from the start. An unexplained subphrenic abscess may have arisen in this way.

(iii) TORSION

This is extremely rare and only occurs in those cysts with a peduncle in the process of extrusion from the liver.

Symptoms. The cyst grows with the individual and its bulk is readily accommodated so that symptoms are often absent until complications bring the patient to hospital for the first time. Nevertheless relatively small cysts situated in vital areas such as the porta hepatis may cause compression of neighbouring structures.

(i) PAIN

This is usually absent though large cysts may produce a dragging ache in the liver area. If pain arises it usually reflects a complication. It may be—

(1) Biliary colic, indicating intrabiliary rupture.

(2) Peritonitic or pleuritic when rupture occurs or is threatened into these cavities.

(3) Cramping and severe in the rare cases of torsion.

(4) Dull and aching in deep seated intrahepatic suppuration.

(ii) URTICARIA

May occur at intervals in an otherwise quiescent case and normally marks a minor leakage of hydatid fluid through the cyst wall. The urticaria may be accompanied by a more or less severe generalized anaphylaxis

(iii) Jaundice

Is unusual in uncomplicated cases apart from the cyst located in the porta hepatis. When combined with colic it often is due to hydatid debris obstructing the biliary tree following intrabiliary rupture.

(iv) Gastric Disturbance

Nausea, vomiting, indigestion and postprandial discomfort are relatively common, but sufficiently non-specific to arouse no suspicion of their being due to hydatid disease.

Physical Signs. These will depend on the location of the cyst or cysts. There may be no signs whatever when the upper part of the liver is affected. On the other hand, the liver may be appreciably enlarged and irregularities palpable on the lower edge when, as is usually the case, the lower part of the liver holds the lesion and the hepatic dullness to percussion is continuous over the mass. The cyst is characteristically round, smooth, tense and fluctuant, moving with the liver on respiration. It is rarely pedunculated and lateral mobility is limited. There is infrequently a fluid thrill on percussion over the cyst, analogous to that over the whole abdomen in ascites. A very large cyst may appear to lift from the liver. The liver may encroach upon the chest displacing breath sounds from the lower part of the thorax and on occasion deforming the thoracic cage. There may be tenderness over the mass when infection is present.

Penetrating X-rays may reveal calcification in the liver area (Fig. 57) or gas may outline the cyst perimeter or cavity with a fluid level when anærobic gas forming organisms are present or a communication with the bronchial tree exists (Figs. 58, 59, and 60). More frequently there is an elevation of the right diaphragm which may be rounded or sometimes pedunculated when a cyst is traversing the diaphragm. A lateral view of the chest is helpful in distinguishing a general phrenic elevation from a localized boss. Following rupture into the thorax an effusion and even pleural daughter cysts may be visible in the X-ray. It may not be possible to tell a hepatic cyst from a pulmonary one without resort to pneumoperitoneum when air under the right diaphragm will show whether the cyst is above or below it provided that the subphrenic space is free. Further localization of the mass may be achieved by barium meal, pyelography and cholecystography.

When hydatid disease is suspected the Casoni test may yield confirmatory evidence. This entails the intradermal injection of 0 3 ml. of fresh hydatid fluid (from a sheep) which contains a specific protein and in sensitized individuals causes a local wheal. This is compared with a saline control injection. The size of the wheal is measured and when greater than 23 mm. in diameter is said to be positive. The reading is taken 20 minutes after injection but may not be maximal for 12 hours. It is claimed to be positive in 56 per cent of uncomplicated and in 26 per cent of complicated cysts, though figures higher than this are quoted. False positives occur. It is the easiest of all the immunological tests and seems as accurate as any.

The fæces or pus from a sinus should be searched for hydatid elements and the white cell count shows an eosinophilia in 25 per cent of cases.

Differential Diagnosis. A right sided cyst may be confused with—

(1) Mucocœle of the gall bladder.

(2) Hydronephrosis.



FIG. 57. Lateral X-ray showing calcified, crumpled hydatid of liver. Such a cyst is not necessarily dead (Mr N R Barrett's case.)



FIG. 58 Lateral X-ray showing a fluid level and an uneven lining to an infected cavity associated with a hydatid cyst. A daughter cyst is visible. (Mr. N. R. Barrett's case.)



FIG. 59. X-ray taken with patient lying in right lateral position showing a large hydatid cavity into which both air and lipiodol have been injected before its nature was realized. (Mr N. R Barrett's case)

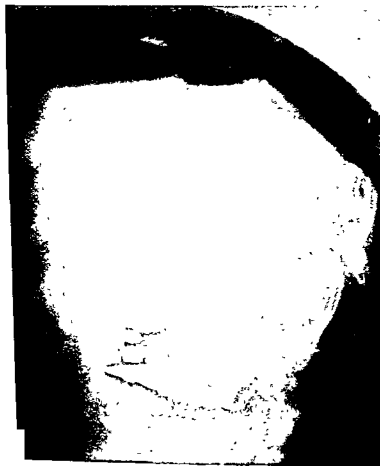


FIG 60. Same patient erect.

(3) Pancreatic cyst.

(4) Degenerating hepatic or renal neoplasm.

In complicated cases many other diseases are imitated. Underlying hydatid disease may present as biliary colic, jaundice, subphrenic abscess, empyema, lung abscess or an acute abdomen. The hydatid basis may not at first be suspected and accounts for much unavailing treatment (Fig. 61).

Treatment

PROPHYLACTIC

This concerns general measures on a national basis such as the disposal of offal beyond reach of any dog, the feeding of dogs with cooked meat and the discouragement of canine pets in affected regions.

THE UNCOMPLICATED HEPATIC CYST

When diagnosed they should be removed as the advent of some complication is most likely and the condition then becomes more dangerous and less curable.

Variable degrees of difficulty are encountered, depending on the site and number of cysts present.

It is vital to remove the cyst whole as only in this way is dissemination of the contents certainly avoided. This is not always possible and certain risks may have to be taken. If there is to be spillage the patient will be better off without operation.

Every effort is made with the assistance of X-rays to locate the exact site and number of cysts present, though available information is often incomplete.

OPERATION. A convenient approach is made:

For the lower cysts by a paramedian or Kocher incision.

For the lateral midzone cysts by a low intercostal incision through the costal margin, with or without opening the pleura.

For the superior ones a transpleural approach incising the diaphragm may give the best exposure.

The liver is then examined.

If the cyst lies within a thin flange of liver or is pedunculated at the liver edge it is best removed by cutting through hepatic tissue and excising the cyst along with its bed. In this way there is no risk of spillage, but the method is only rarely applicable.

More usually the cyst is deeply imbedded in the liver bulk but reaches the surface at one point showing as a whitish patch of varying size. It is rarely possible to enucleate these and the process of "Hydatid birth" successful in the lung is not applicable to the liver. Attempts to destroy the live hydatid elements are necessary and a 10 per cent formalin solution is injected after aspiration of the fluid contents—the wound being well packed with black towels to catch any spillage, which is thus made visible. After a lapse of about 8 minutes the contents are again aspirated by suction pump. The formalin can only be effective in univesicular cysts as daughter cysts are not penetrated unless punctured. The laminated layer is not adherent and can be separated from the host tissues completely and lifted from the wound. The danger of contamination is always present and gloves and instruments which have come in contact with the interior of the cyst

should be discarded. A further swabbing with formalin is carried out and the cavity inspected to make sure that removal has been complete and no "diverticulæ" exist.

The residual space may be large and if not calcified there is an immediate contraction, but a considerable dead space remains. It may fill with bile or blood or pus according to the state of the cyst at operation. There is a danger that this may leak into the peritoneal cavity. In infected cysts, or in the presence of a biliary leak, drainage is



FIG 61. Injection of lipiodol into a draining subphrenic abscess due to complicated hydatid disease of liver The abscess communicates with the biliary tree. The right hepatic duct, common duct and gall bladder are demonstrated Such a cyst probably first ruptured into the bile duct and then, becoming infected, involved the subphrenic space

essential but should not traverse the open pleura. Otherwise the cavity can be safely closed by suture or filled with normal saline to allow slow absorption and contraction of the cavity. In any event it will do no harm and is safer to place a drain down to the area.

The Complicated Cyst

SUPPURATION. Here the problems are essentially those of liver abscess and in fact an underlying hydatid basis may not be suspected. A suppurating hydatid is not necessarily a dead one.

INTRABILIARY RUPTURE. The cyst requires attention as in uncomplicated cases although if there is associated obstruction of the common bile duct by hydatid debris a biliary fistula is a possibility. Choledochotomy and clearance of the ducts with T-tube drainage may be required. Such a complicated cyst may have associated daughter cysts outside the main capsule and they should not be overlooked.

INTRATHORACIC RUPTURE. This requires excision of the cyst and drainage of the cavity through an avenue other than the chest in addition to toilet of the pleural cavity. A resulting empyema is in effect a dumb-bell abscess with connecting cavities in liver and pleura. The empyema is drained in the usual way and the hepatic cavity cleared through an enlargement of the existing track, though the eventual appearance of secondary cysts in the pleura should be anticipated.

INTRAPERITONEAL RUPTURE. If the condition is found the cyst remnants and any recognizable debris and bile are removed as far as is possible, and the hepatic area drained in case of bile leakage.

Secondary cysts of the peritoneum may appear in great numbers after about 5 years and cause surprisingly little disturbance.

Alveolar Hydatids

This rare type of disease is thought to be due to a different tænia occurring geographically where the ordinary hydatid is rarely found. In this form of cyst the daughter cysts are formed on the outer surface and thus burrow like a growth into surrounding tissues. The prognosis is on this account worse and the disease is virtually untreatable.

References

(1) Arce, J. (1941) *Arch Surg.* 42, 1.
(2) Barrett, L. (1944) *Med. J. Aust.* 2, 511.
(3) Barrett, N R. and Dillwyn Thomas. (1953) *Brit. J. Surg.* 40, 222.
(4) Carrodus, A. L. (1935) *Med. J. Aust.* 2, 714.
(5) Dew, H (1930) *Brit. J. Surg.* 18, 275.
(6) Fairley, K. D. (1929) *Med J. Aust* 1, 472.

TUMOURS OF THE LIVER

Benign. Benign tumours of the liver are extremely rare and may arise from any of a variety of cells. They include

(1) Adenoma. (*a*) Of the liver cell.
(*b*) Of the bile duct cell.
(2) Fibroma, myxoma, and leiomyoma.
(3) Hæmangioma, hæmangio-endothelioma and lymphangioma.
(4) Cysts. (*a*) Single true cyst.
(*b*) Multiple true cysts
sometimes associated with cystic kidneys, spleen, and pancreas.
(*c*) Degenerative cyst formation in other tumours.
(5) Mixed cell tumours ranging from hamartoma to teratoma.

The last three categories (with the exception of 4*c*) may represent developmental errors rather than true tumours.

The hæmangioma is encountered most frequently, and in common with other tumours is prone to the complications of hæmorrhage, sepsis, calcification or torsion when pedunculated. They are all of clinical significance only in so far as they may be mistaken for secondary carcinoma or hydatid disease. They are seldom large and although resection is feasible they are probably better left alone when found incidentally. If pedunculated or complicated or unquestionably causing symptoms resection may be considered. The risk of eventual malignant change is unknown though it undoubtedly occurs, especially in the adenomas.

Malignant

(i) Primary Hepatoma

The incidence of primary carcinoma of the liver varies enormously in different parts of the world. The Bantu races of South Africa are particularly susceptible and there is an increased incidence in the Orient. This is partly connected with the distribution of nutritional cirrhosis of the liver. This is not the only factor, however, and racial predisposition, parasitic infestation, and diet seem also to be significant.

In the most susceptible races it is the commonest form of tumour.

There are two main types:

(i) A liver cell tumour—hepatoma—is associated with portal cirrhosis in about 85 per cent of cases. The remaining 15 per cent occur for the most part in children and seem to be a different entity unassociated with cirrhosis. Hæmochromatosis is equally carcinogenic.

(ii) A bile duct cell tumour—cholangioma—associated with cirrhosis in about 50 per cent of cases. The cirrhosis is more often biliary in type.

Clinically the disease is rapidly progressive with anæmia, fever, weakness, cachexia, jaundice, epigastric pain, and mass as the prominent features. These symptoms may have been added to those of a pre-existing cirrhosis and portal hypertension. The tumour occasionally ruptures giving rise to an "acute abdomen" with internal hæmorrhage.

Treatment. The literature contains several examples of successful resection of these growths at a time when surgical facilities were much less developed than at present. While operable cases are few even in so rare a disease the possibility of resection should be kept in mind.

It is only to be contemplated in the case of a single localized tumour where a clear margin of normal tissue can also be removed. Radiotherapy is ineffective. (viz Section III, p. 229—"Partial Resection of the Liver")

(ii) Sarcomata

Sarcomata are even more rare but have been described.

(iii) Secondary Carcinoma

Secondary malignancy of the liver is infinitely more common than primary, and may reach the liver:

(*a*) Through the portal vein.

(*b*) Through the hepatic artery.

(*c*) Through lymphatics.

(*d*) By direct spread.

Most primaries are found in portal territory, particularly in the stomach, colon, rectum, pancreas, and œsophagus, and metastasise to the liver through the portal vein. The hepatic artery is thought to carry malignant cells when a carcinoma of the lung or a melanoma of the eye metastasises to the liver. Breast carcinoma typically reaches the liver by lymphatics running in the falciform ligament, while a gastric or gall bladder malignancy may involve the liver by direct spread.

Almost any malignant tumour may metastasise to the liver though it is uncommon for the squamous lesions of the head and neck, and bone or other sarcomata to do so.

CLINICAL FEATURES. The deposits in the liver are typically though not invariably multiple and distributed throughout its substance. They frequently necrose centrally giving rise to the umbilicated surface and serosal reaction. They are, when palpable, felt as hard bosses inseparable from the liver, which is generally enlarged. They move on respiration and often give rise to a peritoneal friction rub.

They may by their bulk produce secondary effects such as obstructive jaundice or portal hypertension. Ascites can result from peritoneal involvement, while rupture may cause intraperitoneal hæmorrhage or tumour dissemination.

Anæmia, fever, cachexia, and weakness are the eventual symptoms as the liver is progressively destroyed, but such a manner of death is often less unpleasant than the pain and obstruction caused by the untreated primary lesion. This is the argument for the palliative resection of primary tumours, particularly in stomach and rectum.

It is not unusual to be unable to discover any primary growth at laparotomy in the presence of liver metastases.

TREATMENT. The presence of liver secondaries is taken to mean that the disease is for the present incurable The resection of apparently solitary deposits has many times been reported, but the presence of deep seated metastases can never be excluded. The single resectable deposit is very rarely found, but when it is its removal should be attempted as in this abides the only hope of cure, however forlorn.

There is slightly more hope when the liver is involved by direct spread, when a segment of liver can be removed along with the primary lesion. This is especially the case in carcinoma of the gall bladder, though the outlook is bad.

References

(1) Berman, C. (1944) *Clin Proc. Cape Town* **3**, 323.
(2) Clagett, O. T. and Hawkins, W. J. (1946) *Ann. Surg.* **123**, 111.
(3) Dansie, C. B. (1922) *Lancet*, **2**, 228.
(4) [illegible] *lers & Co*, Philadelphia.
(5) [illegible]
(6) [illegible]
(7) [illegible]
(8) [illegible]
(9) [illegible]

SECTION II

THE GALL BLADDER AND BILE DUCTS

CHOLECYSTITIS

Acute Cholecystitis

Ætiology. Acute inflammation of the gall bladder may arise in a previously normal organ or be superimposed upon a pre-existing chronic inflammatory state. In most cases (90 per cent) there is a recognizable obstruction of the outlet, which is usually a stone, and this is referred to as obstructive or calculous cholecystitis as opposed to non-obstructive cholecystitis.

It is believed that in almost all cases of cholecystitis bacteria are concerned at least in part with the inflammatory process, though they may be hard to find. At times they can be found in the bile, at times in the gall bladder wall, or in the cholecystic gland, but in about 20 per cent of all acute cases no organism is recovered, though this does not necessarily mean that none have been present. This fact, together with a microscopic picture in which polymorphonuclear cells are sometimes very scanty, and the frequent harmless presence of organisms in the bile of a healthy gall bladder suggests that other factors are involved. These may be:

(1) Mucosal abrasion by a stone.

(2) Tension within the gall bladder due to an impacted stone causing devitalization of the wall from interference with the blood supply.

(3) Alteration in the bile which becomes injurious as a result of the bile salt concentration. Such a change can be induced experimentally by stagnation or pancreatic reflux and acute cholecystitis results.

All such changes make the gall bladder vulnerable to bacterial invasion of the wall and inflammation.

Route of Infection. Organisms can reach the gall bladder by several channels.

(1) THE BILE. It is thought that organisms constantly reach the liver from the intestine in the portal blood which are there either destroyed or transferred in the bile back to the intestine. Such organisms find their way in and out of the gall bladder and can become dangerous when circumstances allow. This seems the most important route.

The older notion of organisms ascending the common duct from the intestine is no longer favoured.

(2) THE BLOOD STREAM. This avenue is probably of importance in the cases of non-obstructive cholecystitis which may follow infections of the respiratory tract and elsewhere, or in frankly septicæmic states.

(3) LYMPHATICS. It has been suggested that lymphatics may carry the organisms from the liver or intestine, though adequate evidence is lacking.

(4) DIRECT SPREAD FROM THE LIVER. This has been suggested by the frequent adjoining hepatitis. The idea seems improbable.

Bacteriology. The striking feature is the frequent finding of one or more intestinal organisms. B-coli is the most common, but streptococci fæcalis, staphylococci and Clostridium Welchii are also found. These findings are strong evidence in favour of a

fairly direct avenue of infection from the intestine. Typhoid bacilli are now rarely found though they may be present at the time of or long after an attack of enteric.

In non-calculous cholecystitis when the infection is embolic or bacteræmic from another site of disease the organism is not typically intestinal and the subsequent natural history of the infection differs accordingly from the more usual type in that the element of obstruction is not always present, the attack is more acute, more prone to gangrene, and carries a worse prognosis.

Morbid Anatomy. The disease has often been divided into types, according to the severity of the inflammation, which are now recognized more as stages in the evolution of the pathological process which, of course, may resolve at any point. In an advanced case the walls are extremely œdematous and may carry the fibrosis of previous disease. There is much hyperæmia of the serosal surface and hæmorrhage throughout the mucosal and other coats. The mucous membrane may be gangrenous and detached over large areas. A stone is usually impacted in the neck or cystic duct and the gall bladder may contain other stones. The gall bladder content may at first be cloudy bile later becoming frankly purulent. The serosal coat is red and roughened with adherent fibrin. The surrounding viscera and omenta may also be loosely adherent.



FIG 62. Cross section of a chronically inflamed gall bladder in which stones are actually lying within the thickness of the wall outside the mucosa both at the fundus and in the middle zone Some cavities can be seen from which stones have fallen during preparation of the specimen Mixed stones have been removed from the lumen with the exception of the cholesterol one in Hartmann's pouch Such findings give evidence of previous perforation during an acute cholecystitis—a common occurrence.

The attack is thought to start in some such way as this. A gall stone lying in the neck of the gall bladder perhaps causes some local mucosal abrasion and a superficial infection leads to œdema which, with the stone, effectively seals the exit. The secretions accumulate within the gall bladder and lead to considerable tension within the lumen which first impedes the venous and lymphatic circulation giving rise to an œdema and transudation into the lumen, thus further increasing the tension until the arterial supply is impaired and the organisms which are present can spread easily through the devitalized tissues. Gangrene of the mucosa is common; but of the muscle wall only a small patch at the extreme fundus, most distant from the arterial supply (and where the arteries are wholly intramural) becomes gangrenous and gives way under the pressure. Tension is immediately relieved and blood flows back into the organ, being extravasated where damage is severe, but clearing up the infection in due course. On occasions the cystic artery thromboses and the gall bladder becomes totally gangrenous, or an overwhelming infection with Clostridium Welchii produces a similar result.

Meanwhile the serosal inflammation has produced adhesions to the omentum and neighbouring viscera and it is into this that a localized perforation takes place so that general peritonitis is uncommon and in fact there may

be no clinical evidence of the perforation. That it has taken place is proved by the not uncommon finding at a later date of stones lying just outside the fundus of the gall bladder in fibrous tissue over which the mucosa has healed (Fig. 62).

The œdematous adherent viscera and omentum around the acutely inflamed gall bladder from the palpable mass and in this an abscess may form. This may rupture into neighbouring viscera or be incised, and in this way an internal or external biliary fistula results. Should it burst into the general peritoneal cavity peritonitis will develop which may be biliary. Fistulæ will heal spontaneously where a free passage to the duodenum down the bile ducts is re-established. An acute inflammation frequently subsides into a chronic one, especially where stones remain.

In the acute attack many writers stress the discrepancy between physical signs and the severity of the underlying pathology and quote cases where gangrene has been found when the clinical features were subsiding. Few realize that in many cases it is because of the local gangrene and localized perforation which decompresses the gall bladder that the process of resolution can begin.

Complications. The complications of acute cholecystitis may thus be listed:

(1) Perforation.
(2) Peritonitis—septic.
(3) Peritonitis—biliary.
(4) External biliary fistula.
(5) External mucus fistula.
(6) Internal biliary fistula into duodenum, colon, or small bowel.
(7) Ascending cholangitis.
(8) Cholangitic liver abscess.
(9) Obstructive jaundice.
(10) Empyema of gall bladder.
(11) Hepatitis.
(12) Pancreatitis.

(i) PERFORATION

This is much more common than is recognized clinically owing to the surrounding adhesions localizing the burst so that no additional physical signs appear. What matters is whether a general peritonitis results and this occurs in less than 0·5 per cent of all cases.

(ii) PERITONITIS—SEPTIC, BILIARY; BILIARY FISTULÆ; MUCOUS FISTULÆ

The type of peritonitis depends on whether pus or pus and bile are released into the peritoneal cavity. In the latter case the organisms and irritant biliary constituents combine to cause a particularly lethal form of inflammation. The absorption of large quantities of bile salts may contribute to a fatality in which circulatory failure is a common feature.

In the very great majority the peritonitis remains entirely limited to the right upper quadrant where an abscess may form. Such an abscess usually remains local and disperses spontaneously but may either point and be incised through the skin or burst internally into a contiguous hollow viscus. In either event a temporary fistula may result, external or internal. There is a strong tendency to healing and if the fistula persists after about fourteen days there is evidence of an obstruction somewhere in the biliary tract. If the surface discharge is mucous the cystic duct is obstructed, if bilious the

common duct is involved. A permanent fistula into colon or lower ileum may be serious eventually as there may be some impairment of fat, calcium and vitamin absorption, diarrhœa, and excessive sodium loss. Any form of permanent internal biliary fistula is the resultant of the natural healing power and the pressure of bile seeking a passage. Such a compromise is always unsatisfactory and a form of chronic biliary obstruction prevails.

Occasionally a pericholecystic abscess will find the subphrenic space or track even further afield. It may be visible radiologically owing to the high calcium content of the stagnant bile within the abscess cavity (Fig. 63).



FIG 63 Straight X-ray of the gall bladder region showing bile of high calcium content lying within a pericholecystic abscess into which perforation has taken place

(iii) Ascending Cholangitis; Liver Abscess; Jaundice

During the acute attack of cholecystitis organisms are frequently found in the common bile duct and although usually they do no great damage here, on occasions, particularly if there is an associated common bile duct obstruction, a fulminating infection ensues with considerable constitutional upset. The bile is turbid and often purulent and the walls of the bile ducts œdematous and hæmorrhagic. This in itself may suffice to close the finer channels so that jaundice results. In severe cases multiple liver abscess (q.v.) are formed and constitute a very serious complication.

In many acute cases without obvious signs of cholangitis the liver is swollen and impaired function can be demonstrated biochemically in almost half the total number. The pancreas less often shares in the surrounding inflammatory process.

Jaundice occurring during acute cholecystitis may be due to:

(1) Ascending cholangitis.
(2) A stone in the gall bladder neck pressing or ulcerating into the common hepatic duct.
(3) Œdema and inflammatory pressure upon the common duct.
(4) An associated stone in the common bile duct with superadded infection of the wall.
(5) An associated pancreatitis.

Empyema of the gall bladder is the traditional term applied when the organ distends with pus. It carries no special significance.

Clinical Features. Acute cholecystitis may arise suddenly for the first time though there is often a history of similar attacks in the past. In addition many patients have suffered the symptoms of chronic cholecystitis and may have experienced biliary colic or obstructive jaundice previously. The disease is some three times more common in females and the average age is between 45 and 55 though it may occur in youth. The patients are usually obese.

Symptoms

The attack often begins during the night with biliary colic of a mounting intensity and with little intermission of the pain. It is typically epigastric with symmetrical radiation to each costal margin and scapular areas. This phase may last for several hours and corresponds with the obstructive pre-inflammatory stage of the disease. It is accompanied by severe nausea, reflex vomiting and restlessness. The inflammatory phase soon follows and when the serosal coat is reached, localized peritonitic symptoms develop. The pain becomes more definitely located to the right costal margin with radiation to the right shoulder point. This pain is aggravated by movement and deep breathing and is associated with local tenderness beneath the right costal margin. The temperature may rise sharply perhaps to 104 degrees and be associated with rigors. An obstructive type of jaundice is found in about 20 per cent of patients.

Physical Signs

The patient is usually flushed and perspiring and the abdomen moves little with respiration. There is tenderness and moderate rigidity which is maximal in the right upper quadrant, and an indefinite mass or deep resistance may be palpable in the minority according to the length of history. The mass when present lies close under the anterior abdominal wall at the right costal margin and does not lift from the loin. The movement with respiration is not marked, though the tenderness may be exaggerated by inspiration.

At a later stage the mass may exceptionally be recognizably fluctuant or even show signs of pointing. There is rarely any sign of generalized peritonitis. The urine may contain bile pigments and acetone bodies and the conjunctiva be icteric when jaundice is present.

The moderate leucocytosis is of little diagnostic help. Radiological evidence of gall stones may be obtainable if they are mixed in type. Cholecystitis emphysematosa is the name applied to those cases in which the inflamed gall bladder contains gas. This may be due to a virulent Welch infection or a fistula into the intestine.

All degrees of severity of infection are encountered and few cases will exhibit all the above mentioned features. The majority of cases are mild.

Diagnosis. The list of diseases which have been confused with acute cholecystitis is almost infinite. The more commonly encountered liable to lead to confusion include:

(1) A leaking duodenal ulcer.
(2) Right sided pleurisy and pneumonia.
(3) Acute pancreatitis.
(4) Acute virus hepatitis.
(5) Acute pyelitis.
(6) Coronary thrombosis.

Treatment. The ideal treatment for acute cholecystitis is cholecystectomy though the timing of it is still not standardized. The gall bladder should be removed because recurrent attacks are likely and chronic inflammation persists in most cases. Surgeons are divided as to whether operation should be carried out immediately or later, after two or three months, when acute inflammation has subsided. The argument hinges on mortality and the frequency of gangrene, perforation and peritonitis. Under conservative

treatment gangrene has been estimated to occur in from 12–45 per cent of acute cases, but this is not so important as the incidence of general peritonitis as the usual localized patch of gangrene merely ruptures into preformed adhesions and remains contained. General peritonitis probably does not occur in more than 0·5 per cent of acute cases. Figures will vary according to whether mucosal or total gangrene is the criterion or whether contained or uncontained perforation is referred to or whether the state of the gall bladder is judged by clinical signs or operative findings. Furthermore all statistical analyses are influenced by what cases are included under the diagnosis of acute cholecystitis. In this country there are very many mild ones in whom such a diagnosis is no more than an opinion and quite unproven. To include all these in a series is to render the whole enquiry unscientific and the question remains as to how acute and certain a case must be before being accepted for statistical purposes.

Conservative Treatment and Late Operation

This recognizes that in some 90 per cent of all acute cholecystitis patients the inflammation will gradually subside without complications and the mortality of such a method is probably no greater than that obtained by early surgery. The mortality for surgery within the first 48 hours (3·5 per cent) does not include the more seriously ill patients first seen after this time, who may be refused operation and who go to load the mortality figures for conservative treatment (10 per cent). The gall bladder should be removed after an interval of 8–12 weeks when the acute inflammation has settled.

The exponents of the conservative regime argue:

(1) General peritonitis is rare. Local gangrene and perforations are of much less importance.

(2) The acute operation is apt to be hæmorrhagic and therefore less precise with regard to the avoidance of vital structures in the porta hepatis.

(3) That if it is accepted that the common duct should be explored in about one in three operations for chronic cholecystitis, it will probably also be required only slightly less frequently in acute cases. Such a manœuvre, with the handling of duodenum and pancreas and perhaps retroperitoneal exposure is dangerous in the presence of acute inflammation and is seldom done. Nor is the palpation of common duct stones easy under these circumstances. At operation in the cold state a full choledochotomy routine can be safely followed where indicated.

(4) The recent use of antibiotics (in selected cases) has further reduced the seriousness of the disease.

(5) The patients are often old and fat and with poor hearts and should not be subjected to risk under unfavourable conditions of fever, virulent sepsis, dehydration, acidosis, hepatitis, etc.

(6) An abscess may already have formed and the infection be effectively walled off, only to be disseminated by the laparotomy.

(7) Even with early surgery it occasionally happens that a cholecystostomy is all that is possible and a further operation will still be required.

Conservative therapy includes:

(i) Rest in bed. A Fowler's position is no longer recommended.

(ii) The use of an unrestricted fluid diet containing plenty of sugar, with an early return to light solids as soon as improvement becomes established.

(iii) The use of pain relieving drugs such as pethidine and morphia is not recommended in biliary surgery from their reputation for causing spasm of the sphincter of Oddi.

(iv) The occasional use of intravenous glucose and perhaps even saline where uncontrolled vomiting is leading to dehydration and hypochloræmia.

(v) Antibiotics may be used, but the majority of cases will settle very adequately without them. They should be used only when indicated:

(*a*) In the presence of cholangitis or jaundice.

(*b*) In a fulminating attack.

(*c*) In the bad risk patient who fails to improve.

The antibiotics must be effective against gram negative organisms and a combination of streptomycin or terramycin and penicillin copes with the likely bacteria.

Conservative therapy is abandoned in the presence of deterioration as exhibited by spreading peritonitis, increasing jaundice or the pointing of an abscess. Under these circumstances the gall bladder is drained after the removal of stones where cholecystectomy seems too hazardous. In very sick patients a simple incision and drainage of the abscess may have to suffice.

Radical Treatment

The advocates of immediate operation usually specify that a few hours may be spent in the improvement of the patient's general condition by careful intravenous glucose and saline and the use of antibiotics. They argue that:

(1) The list of complications of acute cholecystitis is formidable and they are dangerous; they are mostly avoided by early operation.

(2) Perforation and gangrene are common.

(3) The mortality of early operation is less than the combined mortality of conservative therapy and late cholecystectomy.

(4) The patient's time and the hospital bed space are saved by a single admission to hospital.

(5) In cases treated conservatively a few will deteriorate and require urgent surgical intervention. These are the dangerous ones which could have been saved from such a risk had early operation been carried out.

(6) The operation is easier when œdema separates the tissue planes and before fibrosis obliterates them.

(7) Some patients will develop another attack between the first and the time planned for the cold operation.

The exponents of early operation will nevertheless usually adopt conservative measures if the attack has been running for two or three days and especially if clinical improvement is already manifest in a patient who presents a very bad operative risk.

Chronic Cholecystitis

Chronic cholecystitis is divided into two groups according to whether stones are present. They are spoken of as calculous and non-calculous cholecystitis and are for the most part clinically indistinguishable in the absence of jaundice. Chronic gall bladder disease is common and is found in some degree in about 68 per cent of all adult necropsies.

Ætiology. Many cases represent the aftermath of an acute cholecystitis in which the gall bladder wall has never returned to a normal state but has been left fibrotic and maimed. In other cases the condition appears to have arisen gradually without a preliminary acute attack. Stones are present in about 80 per cent of true cases coming to surgery and appear to have antedated the infection in most instances.

Much discussion about the role of bacteria in the ætiology has arisen from the failure to obtain a positive culture from bile, or bladder wall or cholecystic glands in about half the cases, also the difficulty in reproducing the disease by organisms alone. When they are found the bacteriology does not differ materially from that of acute cholecystitis.



Fig. 64. Chron[illegible]

The most convincing explanation is that a partial obstruction of the cystic duct exists and may be due to either stone, stricture, fold, kink, or valve. Such a partial obstruction brought about experimentally reproduces the disease in dogs. It may act by favouring the retention of a toxic over-concentration of bile salts in the gall bladder—a concentration only twice that of normal having been shown to damage the mucous lining. The invasion of organisms may thus be a secondary factor.

Cholesterosis of the gall bladder nearly always shows an associated chronic inflammation.

Pathology. The chronically inflamed gall bladder is thick walled and often contracted down onto the stones contained within it (Fig. 64). The wall may be slightly œdematous and the normal transparent blue appearance gives place to an opaque dull yellow surface upon which adhesions are frequently found. Sometimes these are filmy and look almost like anatomical peritoneal folds connecting the gall bladder waist with duodenum or colon. At other times they are extremely dense with matting of all the adjacent viscera In such a state an internal fistula is sometimes found. The adjacent liver shares in the fibrotic process. Fibrosis distorts the gall bladder and causes narrowing especially at the [illegible] osited [illegible] The mucous membrane is injected and thickened and may be ulcerated or even polypoid or cystic in places. Microscopically fibrosis of all coats and infiltration with chronic inflammatory cells are evident; the mucosa is at times lacking and occasionally sending penetrating gland-like processes into the depths of the muscular wall (sinuses of Rokitansky)

which may be mistaken for carcinomatous infiltration. In severe cases mucosa, muscle and serosa may be hard to find, and small abscesses punctuate the section. There may be actual cholesterosis of the gall bladder and the cholecystic gland is usually enlarged.

Complications. These are similar to those outlined under the section on gall stones.

Clinical Features. Not every patient with a mildly inflamed gall bladder is necessarily aware of it and conversely many vague symptoms are erroneously ascribed to minimal changes in the gall bladder. The disease is twice as frequent in women.

Symptoms

Flatulent Dyspepsia. This is the most usual symptom and consists of an epigastric discomfort soon after meals, particularly large and fatty ones. The sensation is one of bloating and distension accompanied by much ærophagy in the attempt to bring up what feels like wind. This indisposition occurs irregularly over long periods of time and lacks the periodicity of peptic ulceration.

Pain. At times the discomfort amounts to a definite central epigastric or subcostal pain which may be knife like or pressing and may pass through to the scapular region. Such a pain may be severe enough to be indistinguishable from biliary colic. Nausea is a very frequent accompaniment even in mild disease and there may be some vomiting and heartburn.

These symptoms may at times be stepped up to a more disabling intensity and a state of subacute cholecystitis supervenes.

At any time one or more of the symptoms due to stone, such as obstructive jaundice or true colic, may be added and constitute an important aid to diagnosis.

Cardiac Symptoms. There seems to be an ill defined correlation between chronic cholecystitis and the state of the coronary arteries. At times the gall bladder symptoms appear to mimic myocardial insufficiency, but at others a true angina or dyspnœa of effort may be relieved by cholecystectomy. It may be that the cholesterol deposits of coronary atheroma are influenced by the disturbance of cholesterol turnover of gall bladder disease.

In many patients the symptoms are non-specific but gall bladder disease is revealed during the course of investigation of atypical abdominal complaints.

Physical Signs

These patients are commonly obese and over the age of forty. There may be tenderness beneath the right costal margin particularly at the height of inspiration (Murphy's sign). The gall bladder and its stones may be palpable as a hard area at the liver edge and a watch should be kept for signs of jaundice or choluria. Most of the weight of diagnosis depends on radiographic investigation. A straight X-ray of the gall bladder area may show calcium-containing stones which are usually of the ring type. A radio-paque or mixed stone carries a significance indicating past or present infection which is not implied by the calcium free stone. Calcification of the gall bladder wall is rarely seen. Calcified costal cartilages are occasionally mistaken for stones. A lateral view is helpful, especially in distinguishing renal calculi which lie posteriorly and in contrast

to gall stones are usually homogeneous. In a gall bladder obstructed at the neck the contained bile may increase its calcium content to the point of being visible on X-rays—the limey bile of radiologists (Fig. 65).

Cholecystography. This depends on the taking of a radiopaque substance, such as Telepagne, which is absorbed from the intestine, excreted by the liver, and concentrated in the gall bladder where it is opaque to X-rays. The radiological appearance is thus vitiated by failure to take the dye, by vomiting and by diarrhœa, which it sometimes causes. These factors may be checked by the presence of the dye in the ascending colon where it should be visible in the sixteen hour films. The excretion from blood to bile is impaired in diseases of the liver cell and for this reason and for the fear of aggravating the condition the test is rarely used in cases of jaundice or liver failure. In the bile the dye is not normally visible on the film unless it is concentrated by the gall bladder. For this reason the test is useless as a method of visualizing the common duct after cholecystectomy. Failure to concentrate may be due to a blockage of the cystic duct preventing the dye entering the gall bladder or to a diseased mucosa which can no longer absorb. A diseased mucous membrane may nevertheless concentrate on occasions. A faint concentration may indicate a partial failure. A small percentage of normal gall bladders fail to show on occasions. The muscle of the wall is tested by the administration of a fatty meal which in the normal causes gall bladder contraction. When fibrosis replaces or impedes the contraction no alteration in size takes place. This part of the test can, of course, only apply when dye is actually visible. Failure to empty is not a proof of cholecystitis. The cholecystogram is most helpful in revealing stones which, containing no calcium, are invisible on the straight X-ray. (Figs. 66 and 67.) When dye is present in the gall bladder they may show as negative shadows, especially after contraction of the gall bladder. The demonstration of stones is partial confirmation of a suspected chronic cholecystitis



FIG 65 Straight X-ray of gall bladder showing bile of high calcium content in which flocculation has occurred Such bile is said to be found in gall bladders in which the cystic duct is completely blocked This gall bladder contained only one stone, which was impacted in the cystic duct

In equivocal cases, especially when a faint shadow has shown, it is sometimes worth while repeating the examination after an interval with a larger dose of dye, when in many instances a normal cholecystogram is obtained. The dye can be administered intravenously when the oral route upsets the patient or is contraindicated, as in gastrointestinal disease.

With a barium meal the duodenum may be deformed in the presence of chronic

cholecystitis. This appearance is likely to be erroneously taken for evidence of a duodenal ulcer.

The use of biligrafin as a method of visualizing the common duct is described under common duct stones.

Diagnosis. Chronic cholecystitis should not be held to explain symptoms without strict criteria for such a diagnosis. Tenderness under the right costal margin is not sufficient evidence, nor in the cholecystogram is poor concentration or failure to empty



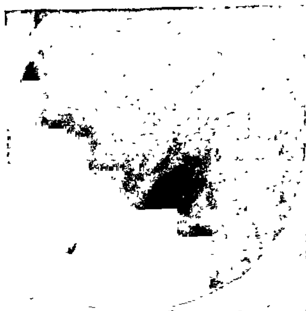


FIG. 66. [illegible] esterol only. The concentration is good. The cystic duct is unobstructed

FIG. 67. Cholecystogram of a good functioning gall bladder containing two mulberry pure cholesterol stones. The gall bladder is almost certainly healthy and thin walled. It is in such cases that the operation of cholecystotomy requires further evaluation. The dye is visible in the ascending colon at 16 hours.

a certain proof. A gall bladder that concentrates normally and empties is highly unlikely to be the cause of symptoms in the absence of stones. Nevertheless it may not always [illegible]

[illegible] will gradually show their hand. When mild obstructive jaundice accompanies an attack, when gall stones are visible and there is no concentration of dye, when the distribution and type of pain are typical and dyspepsia is flatulent, there is no room for doubt.

The disease is most likely to be confused with:

(1) Functional dyspepsia.
(2) Peptic ulcer.
(3) Chronic pancreatitis.
(4) Recurrent appendicitis.
(5) Hiatus hernia and diseases of the lower œsophagus.
(6) Coronary artery disease.

Treatment. The chronic inflamed gall bladder does not often heal and is an ever present danger to its owner for even though it remains uncomplicated it constitutes a septic focus and a source of discomfort and indigestion. Its greatest menace lies in the frequency of complications, many of which are dangerous and incapacitating. For these reasons and when the diagnosis is not in doubt operation should be advised as there is no comparably effective medical means of treatment.

Operation may be deferred, perhaps indefinitely, when the diagnosis is uncertain and when symptoms are sufficiently trivial or infrequent to justify it, though expectant treatment may carry a certain small risk. In such a patient all that can be done is the avoidance of fatty foods, the reduction of weight and the questionable use of chologogues.

There is an undoubted difference in the results of cholecystectomy when done for calculous cholecystitis as opposed to non-calculous disease. The presence of calculi is associated with a better post-operative result, mainly due to the fact that the gall bladder containing stones is more likely to have been the cause of the preoperative symptoms. The non-calculous specimens include those removed under a mistaken diagnosis, in which the minimal changes so common to middle age are reported by the Pathologist. The removal of gall bladders on the strength of flimsy macroscopical shortcomings at a roving laparotomy is to be condemned as seldom producing a happy patient.

The surgical treatment is cholecystectomy with choledochotomy where indicated (*see* later), for which the overall mortality is less than 1 per cent.

Cholesterosis of the Gall Bladder

In this condition, which is not very common, the mucosa of the gall bladder is dotted with macroscopically visible cholesterol deposits which may throw the mucosal surface up into multiple cholesterol containing polypi. The cholesterol is both intracellular and extracellular and the cause of its appearance is unknown, though hotly debated.

The importance of the condition lies in the associated presence of cholesterol stones in 75 per cent of cases, and also of chronic cholecystitis. It is held to be responsible for the formation of some cholesterol stones by the shedding of these cholesterol polypi and thus providing a nucleus.

It is a questionable cause of symptoms in the absence of stones or chronic inflammation. The diagnosis can hardly be made clinically and scarcely at laparotomy without opening the gall bladder. The question of treatment therefore seldom arises as most instances are provided by postmortem or operative cholecystectomy specimens which have deserved excision on account of stone or chronic inflammation.

References

(1) [illegible]
(2)
(3)
(4)
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(7)
(8)

PLATE III

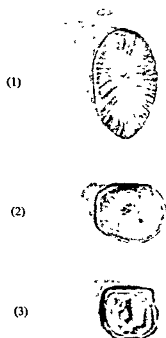


Fig 68 Gall bladder stones
1 Pigment 2 Cholesterol 3 Mixed

CHOLELITHIASIS

Incidence. The frequency of gall stone increases with age until over the age of forty one person in every four is affected and of these the ratio is three or four women to one man. Married women are probably no more susceptible than the unmarried.

Stones are found lying in any part of the biliary tract though the great majority are in the gall bladder where they are formed. Nevertheless stones appear to form in the common duct following cholecystectomy though it can never be certain that a small fragment from the gall bladder has not escaped attention at the time of cholecystectomy.

Common duct stones are present in about 25 per cent of cases coming to cholecystectomy.

Types of Stone. Various types of stone exist, each carrying a different significance. These are (Fig. 68, Plate III):

(1) Pigment stones.
(2) Cholesterol stones.
(3) Mixed stones.
(4) Rare stones.

(i) Pigment Stones

These are composed of bilirubin or occasionally biliverdin and are typically multiple and crumb-like. They are coal black or very dark green or brown in colour and often exist in the common bile duct as well as the gall bladder and no doubt much of this sort of material normally passes through the sphincter of Oddi.

They are normally found in cases of chronic hæmolytic anæmia, where owing to the enhanced rate of red corpuscle destruction the bilirubin turnover of the liver is increased. Under these conditions the pigment is thrown out of solution in an otherwise normal biliary tree. Acholuric jaundice is the most frequent hæmolytic state met with in this connection, though such stones are met with where there is no known predisposing cause. The calcium content is very low and they are not radiopaque and constitute about 5 per cent of all biliary calculi.

(ii) Cholesterol Stones

When pure, such stones may be single (solitaire) or multiple. They are often smooth or mulberry shaped and on section the interior is arranged in crystals radiating from a central point. The single stones often impact in Hartmann's pouch. Such stones are sometimes called metabolic to indicate that they arise not as a result of infection but in response to some alteration in the physico-chemical balance between the various bile constituents.

(iii) Mixed Stones

These are most frequently found and account for some 80 per cent of all biliary calculi. They are sometimes known as infective stones as they are associated with an infected gall bladder in almost all cases. They are usually multiple and faceted due to contact with one another. Sometimes the entire gall bladder is occupied by a single stone or it may be in two or three barrel-shaped pieces. Such stones are *typically* laminated, like a tree trunk, and although mostly formed of cholesterol the laminæ alternate between pigment, calcium and cholesterol and various other substances in small traces.

The calculi when multiple are usually all of a size, or are in schools, grouped according to two or three different sizes corresponding with their age. The calcium content is high (1 per cent) and they are often radiopaque, though of all stones not more than 10–20 per cent will show in a straight film.

A *combination stone* consists of an originally pure cholesterol stone which has more recently acquired a coating of "mixed" composition. This arises in a gall bladder in which inflammation has arisen some time after the formation of a metabolic stone.

(iv) Rare Stones

Calcium carbonate stones occur and are more like urinary calculi.

Ætiology of Gall Stones. Various factors have from time to time been stressed as contributing to gall stone formation but an exact understanding of their causation is still awaited.

(i) Composition of Bile

The gall bladder is a concentrating organ in so far as the bile within it is commonly ten times more concentrated than that leaving the liver. The cholesterol and pigments are thus in danger of precipitation if this process goes too far. The solubility of these substances also depends on the concentration of bile acids and the colloidal content of the bile, both of which are subject to alteration either spontaneously or as the result of infection. It is not known for certain whether cholesterol is secreted or absorbed by the gall bladder mucosa, but derangement in either direction will affect the concentration.

Increase in blood cholesterol occurs in diabetes, pregnancy, myxœdema and other diseases and while the incidence of diabetic stones has been estimated as slightly higher (31 per cent) than in non-diabetics (21 per cent) stone formation has not been shown to be more common in the other diseases. In any case the correlation between blood and bile cholesterol is not known. The influence of pregnancy is highly controversial and while symptoms often date back to a pregnancy the disease is equally frequent in nulliparæ.

(ii) Biliary Stasis

This is a theoretical concept inspired by the known effects of urinary stasis in the ætiology of kidney and bladder stones. So far as the gall bladder is concerned there is no clear cut evidence of stasis other than in known cases of biliary obstruction in which stone formation is not a feature.

In pregnancy the biliary musculature may come under a hormonal influence by which smooth musculature is inhibited. It is claimed that this may favour stone formation.

(iii) The Nucleus Theory

It is known that stones sometimes form around bits of catgut or thread from a previous operation and within some stones can be found a central nucleus differing from the rest of the stone. The nucleus may be clumps of bacteria, or inspissated pus which constitute a basis upon which crystallization proceeds. Similarly the polypoid aggregations of cholesterol in the mucosa of a "strawberry" gall bladder are shed into the lumen and form the beginning of a cholesterol stone.

(iv) Aschoff Theory and Infection

This constitutes the most plausible explanation of the frequent findings in gall bladders filled with mixed stones. The patient starts with a single round cholesterol stone which lodges in the neck of an apparently normal gall bladder. This impacts, obstructing the cystic duct, and initiates an acute cholecystitis. The gall bladder fills with pus but eventually the obstruction relents and bile re-enters mixing with the pus

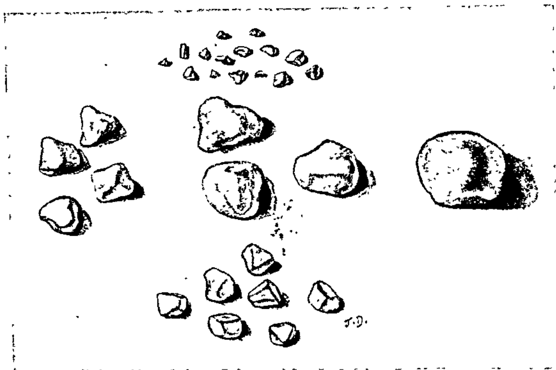


Fig 69. A "school" of stones showing, right, the headmaster and in descending sizes the "boys" of the sixth, fifth and shell forms Finally the lower school, smallest and youngest of all

which inspissates into particles upon which crystallization begins and a brood of stones is born which steadily grow by the deposition of concentric laminæ of pigment, calcium, and cholesterol, as does the original cholesterol stone, which is then known as a combination stone.

More calcium is deposited in an obstructed gall bladder than in other circumstances as is sometimes noticed in a stone lying half in and half out of such a gall bladder when the calcium accumulates upon one end only. A second obstructive episode eventually ensues and another brood is born. So it comes about that a gall bladder contains several broods of stones, each brood being all of an age and size. A school of stones is thus formed (Fig. 69). The headmaster obstructing the exit, holds back the various classes, the older pupils being larger than their more recently entered schoolmates. Such a theory is not accepted as an explanation of all mixed stones for they are sometimes found in gall bladders showing no evidence of previous infection. Not only may the solitary cholesterol stone become in this way a combination stone, but any number of pre-existing cholesterol stones may similarly acquire their first radiopaque shell for the first time when they are quite large.

Only in this sense is infection thought to be instrumental in the formation of gall stones, though the converse is very frequent.

Numerous other factors have been blamed such as a specific stone-forming streptococcus, lack of vitamins, reflux of pancreatic juice, diet, habitus, etc., but their role is of doubtful importance.

Complications. Gall stones are of surgical importance because of the many complications caused by them. They may be listed:

(1) Cholecystitis and sequelæ.
(2) Mucocele of the gall bladder.
(3) Obstructive jaundice.
(4) Cholangitis and liver abscess.
(5) Stricture of the common bile duct and fibrosis of the sphincter of Oddi.
(6) Pancreatitis.
(7) Biliary fistulæ—internal and external.
(8) Intestinal obstruction—"gall stone ileus."
(9) Cardiovascular disease.
(10) Carcinoma of the gall bladder.

(i) Cholecystitis

Roughly 90 per cent of all cases of acute cholecystitis are associated with the presence of gall stones. They may precipitate the attack by obstructing the outlet, leading to considerable intracystic tension, at the same time abrading the mucosa and thus allowing organisms to penetrate into the gall bladder wall. The sequelæ of cholecystitis are considered elsewhere.

The acute phase may pass over into a chronic one and the continued presence of the stones tends to prevent a complete resolution of the acute inflammatory attack. Such acute episodes are repetitive, but sometimes a chronic inflammation becomes established without any recognizable acute attack.

(ii) Mucocele of the Gall Bladder (Fig. 70)

Under certain conditions when bile is prevented by an impacted stone from entering the gall bladder and virulent bacteria are not present the viscus distends with its own mucous secretion, sometimes to a very large size. It is not possible to reproduce this experimentally in a normal animal as a ligature around the cystic duct results in atrophy and gradual contraction of the viscus. Some degree of mild inflammation must also be present for a mucocele to result.

(iii) Obstructive Jaundice

Jaundice accompanies gall bladder disease under a variety of circumstances.

(1) A stone may lie in the common bile duct.

(2) A stone in Hartmann's pouch with surrounding inflammatory induration may obstruct the common bile duct. Such a stone may be in the process of ulcerating into the common duct.

(3) Ascending cholangitis secondary to stones may account for the jaundice.

(4) A common bile duct stricture may result from ulceration due to a stone which has since moved on.

(iv) Ascending Cholangitis

Stones lying in the common duct render it susceptible to recurrent inflammation which still further reduces the lumen causing obstruction where previously none existed. The infection under these circumstances is more dangerous and prone to spread up the entire biliary tree, affecting the fine biliary channels so that a suppurative cholangitis exists and may lead to multiple cholangitic liver abscesses. In longer standing biliary obstruction biliary cirrhosis of the liver is common.

(v) Stricture of the Common Bile Duct

When a stone lies long in the duct recurrent ulceration of the contiguous mucosa may lead to fibrosis which is free to contract when the stone has moved on either through the sphincter or by ulceration into a neighbouring viscus.

Stricturing occurs less frequently than might be expected. So-called obliterative cholangitis does not seem to be connected with the presence of stones. A more frequent finding is a fibrosis and narrowing of the sphincter of Oddi so that a probe is passed only with difficulty and the orifice resists dilatation.

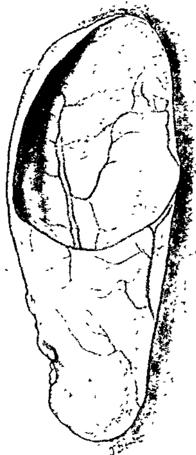


Fig 70. Mucocele of gall bladder. Thin-walled, bluish and transparent, containing colourless mucinous fluid Can be produced experimentally in dogs by a combination of obstruction and a mild degree of infection. (St. Thomas's Hospital Museum.)

(vi) Pancreatitis

Acute pancreatitis is associated with gall stones in two cases out of three and this is understandable where biliary and pancreatic ducts join before entering the duodenum. This anatomical arrangement has been found with widely differing frequency. Some writers quote a figure of 77 per cent and some as low as 3·5 per cent. Closure of the sphincter or an impacted stone could theoretically cause regurgitation of bile up the pancreatic duct, an incident which is known to produce acute pancreatitis. Infected bile is more potent in this respect than is the normal.

(vii) Biliary Fistulæ

When stones exist in a chronically inflamed gall bladder which are impacted and too large to leave the gall bladder they may slowly ulcerate their way through into any viscus which has become adherent to the gall bladder, or a stone may pass first into a pericholecystic abscess and thence into the alimentary canal. The duodenum is most commonly entered in the second part, though colon, stomach or jejunum may be

involved. An external fistula may arise in the same way following the surface drainage of an abscess.

The stone leaves in its wake a fistula which usually heals, but may persist, and while a fistula into the stomach or jejunum is comparatively unimportant, when the colon is concerned the small bowel may receive no bile at all. This can lead to defective fat absorption and all it entails in the form of anæmia, hypovitaminosis, steatorrhœa, etc. Duodenal ulcer and cholangitis may also occur.

(viii) Gall Stone Ileus

A gall stone passing into the alimentary canal may fail to pass through the lower ileum which is the narrowest part of the gut. Obstruction is much less common at the duodenum and the pelvic colon. The patients are usually old and the mortality is high.

(ix) Cardiovascular Disease

This is a most difficult and hotly debated subject. The coexistence of chronic gall bladder infection and coronary artery disease is statistically significant and many cases are on record where apparent angina has been alleviated by cholecystectomy. How much of this is due to some common underlying predisposition and how much to the gall bladder symptoms being confused with those of heart disease it is difficult to say. The impression remains that when coronary artery disease and chronic cholecystitis coexist the heart symptoms will add to rather than subtract from the indications for cholecystectomy.

(x) Carcinoma of the Gall Bladder

Gall stones are present in from 60 per cent to 100 per cent of patients with gall bladder carcinoma though it has been computed that the risk of contracting it are less than 1 per cent when stones are present.

Clinical Features. Gall stones may be quite silent. The symptoms due to stones alone are generally inseparable from those due to the concomitant chronic infection of the gall bladder which are discussed under the section on cholecystitis.

Biliary Colic occurs infrequently independently of any superadded infection and in such cases is seen in its purest form—that is a visceral pain referred segmentally to the parietes as in all other forms of colic. Embryologically the liver grows as a mid line bud from the gut and should therefore acquire a bilateral visceral innervation. Biliary colic is consequently a mid line pain with symmetrical radiation to the back and to either side in the region of the seventh and eighth thoracic segments. That it is commonly more severe on the right than the left is said to be due to the infective element so frequently coexisting which is really providing direct parietal peritoneal stimulation and accounts also for the pain referred to the shoulder tip. This view point is perhaps academic, but serves to align the two sensory components of the pain of obstructive appendicitis with that of cholecystitis and stone.

The timing of the pain differs from other colics in so far as it is less intermittent and may maintain a rising intensity for an hour or two with very little relief. The patient usually sits up in bed and leans forward or is restive during attacks and after the severe pain has gone it leaves the upper abdomen sore for a day or two. Such pain is usually due to a calculus passing through or stuck in the gall bladder neck or cystic duct. It can

be very severe and is accompanied by vomiting and sweating. The colic may initiate the beginning of an attack of inflammation. During and following an attack of colic there is usually some rigidity maximal in the upper right quadrant and some tenderness in the same area.

Almost all other symptoms, with the possible exception of jaundice, are in some measure due to the ineffective element and are considered elsewhere.

Large gall stones completely filling the gall bladder are sometimes palpable at the liver margin. They move with the liver and feel extremely hard. Apart from this uncomplicated stones will rarely reveal their presence except radiographically. Those containing calcium may throw a ring shadow or a cholecystogram show negative filling defects in the gall bladder.

A mucocele is felt as a large, tense, smooth, globular swelling in the right abdomen moving on respiration with the liver, but pedunculated and swinging on a pivot at its superior margin. It is cystic and dull to percussion but does not lift from the loin. It can be confused with any other right sided abdominal cystic swelling.

Treatment. It is often taught that the presence of uncomplicated gall stones is no indication for surgery as they are so commonly asymptomatic. Others argue that the risk and dangers of complications are so great that they, and the gall bladder, should be removed on sight. A policy somewhere between these two extremes is probably more practical and desirable.

When complications arise it is a different matter and the treatment becomes urgent and is dealt with under their appropriate sections.

When stones are found at operation in an otherwise normal looking gall bladder it is sometimes tempting to remove the stones and do no more. But if, as is usually the case, the operation has been required because of gall bladder symptoms it is better to remove the gall bladder in all cases as microscopic disease is often found eventually. The gall bladder may be conserved in those cases (e.g. chronic pancreatitis) where it is felt that it may be required for a short circuit operation at a later date.

The pain of biliary colic is best controlled by injections of Pethidine (100–200 mgms.) and atropine (gr. 1/100). *Morphia is better avoided as it causes contraction of the* sphincter of Oddi and may increase the retching. The application of heat to the area of pain reference is partly effective.

Biliary colic may be severe and frequent enough to demand cholecystectomy, even when no complications exist.

References

(1) Cowley, L. L. and Harkins, H. N. (1943) *Surg. Gynec. Obstet.* 77, 661.
(2) Eliason [illegible] 914.
(3) Este [illegible]
(4) Grat [illegible]
(5) Grat [illegible]
(6) [illegible]
(7) [illegible]
(8) [illegible] *W. B. Saunders* [illegible] Philadelphia.
(9) Wolfson, W. L. and Rothenberg, R. E. (1936) *J. Amer. med. Ass.* 106, 1978.
(10) Zollinger, R. (1935) *J. Amer. med. Ass.* 105, 1647.
(11) Zollinger, R. and Cutler, E. C. (1943) *J. Amer. med. Ass.* 121, 481.

JAUNDICE

An account of the metabolism of bile pigments is beyond the scope of this book and is not here included. The surgeon is concerned with the recognition and differentiation of the various types of jaundice.

The Presence of Jaundice. The recognition of jaundice is not inevitable though the realization of its presence often clinches an otherwise uncertain diagnosis. In a minor degree it will be missed unless specifically looked for. When the skin is stained it is usually obvious, though slight pigmentation of conjunctivæ can often be a matter for argument, particularly in artificial light. In such cases more definite confirmation is required and an examination of the urine for pigments is the first line of enquiry. The presence of bile pigments and acids or urobilinogen in excess confirms the presence of jaundice, though their absence from the urine does not exclude a prehepatic type such as acholuric jaundice. The second line of enquiry concerns the serum, where a bilirubin estimation provides final, accurate confirmation.

The Degree of Jaundice. In subclinical jaundice, although the pigment may be detected in the urine, further quantitative confirmation of the serum pigments may be obtained either by matching the colour of the serum—icteric index, or by a direct measurement of serum bilirubin—quantitative Van den Bergh, which is more accurate, particularly in the higher range. The normal level varies between 0·1–0·8 mgm. per 100 cc. The possession of repeated readings is of value as an index of fluctuation of the clinical state. The icteric index is occasionally vitiated by the presence of other pigment such as carotene.

Differentiation of Types of Jaundice. The three types of jaundice, hæmolytic, hepatocellular and obstructive, all come the surgeon's way and he is not exonerated from having to distinguish between them while selecting those requiring surgery. Unfortunately these types are not always completely distinct as a longstanding obstruction will damage the liver cell, particularly when accompanied by infection. Similarly a degree of obstruction can accompany a primary cellular hepatitis: the finest biliary canaliculi being thought narrowed by cellular swelling and the accumulation of inflammatory products. It follows that there is no single touchstone or combination of them which will surely provide the answer in a given case. Only attention to history, physical signs, and finally laboratory tests will ensure the minimum of error. The surgeon is mainly concerned with the separation of obstructive from all other forms of jaundice.

(i) History

(*a*) Mode of Onset. Jaundice of sudden onset accompanied by dramatic symptoms is suggestive of a mechanical or inflammatory obstruction. A gradual insidious onset occurs more frequently in malignant disease.

(*b*) Previous Attacks. Jaundice due to gall stones is particularly liable to be repetitive, or previous attacks without jaundice may be recognizably similar to the patient.

(*c*) Discoloration of Stools and Urine. This is the main historical point which differentiates many, but not all, cases of surgical jaundice, though the concentrated dark urine of a dehydrated patient is sometimes erroneously accepted as bile-containing. The

hæmolytic processes do not discolour urine or stools (with the exception of hæmoglobinuria, which is frankly red or black and can be differentiated spectroscopically).

(*d*) General Malaise. The patient with severe hepatitis experiences a degree of nausea malaise, anorexia and headache, which is seldom the cases with obstructive jaundice.

(*e*) Family History. Some of the hæmolytic processes are familial and there are rare instances of the members of certain families who normally run at a blood bilirubin level which is frankly icteric (Meulengracht's disease).

(*f*) Anæmia. Is often a feature when chronic or acute hæmolysis has occurred. The patient with pernicious anæmia is recognizably jaundiced. When malignancy is far gone chronic hæmorrhage or hæmolysis may give a low hæmoglobin level, or invasion of bone marrow lead to aplasia.

(*g*) Drugs. Certain drugs, such as chloroform, atophan, and rarely sulphonamides, are toxic to the liver and may cause jaundice; or alternatively an injection or transfusion during the previous three months may be the only clue to a homologous serum jaundice. Mepacrine tinges the skin of those who take it regularly.

(*h*) Occupation. Certain trades carry a risk of liver damage whether by poisoning due to phosphorus, mercury, carbon tetrachloride, dopes, and T.N.T., etc.; or exposure to leptospiral infection.

(*i*) Hæmatemesis and Melæna. The vomiting or passage of blood usually denotes an ulcerating lesion of the alimentary tract, which when associated with jaundice usually means malignancy. Though portal hypertension may similarly lead to gastrointestinal hæmorrhage it is not often that this occurs in the jaundiced patient.

(ii) Physical Signs

The jaundiced patient first invites an examination of the liver.

(*a*) Liver. *Size.* The liver is enlarged in most obstructive states of any severity, and in early hepatocellular disease. It may be impalpable in hæmolytic processes and even shrunken in late cirrhosis.

Surface. A smooth surface is of little help though the presence of palpable knobs is of vital importance. Regeneration nodules of portal cirrhosis may be as large as 2 cm. in diameter, but large knobs are likely to be metastases, especially when umbilicated and associated with a friction rub. A syphilitic hepar lobatum is excessively rare. Hydatid cysts are sometimes recognizably fluctuant or give an hydatid thrill.

Consistency. Metastatic nodules are very hard, as is the cirrhotic liver. The consistency in obstructive and infective disease is less hard though firmer than the normal.

Tenderness. The liver is abnormally tender in hepatitis either due to virus or bacterium and surprisingly insensitive in purely obstructive jaundice.

(*b*) Spleen. An associated splenomegaly is generally indicative of long standing liver pathology though rapid enlargement can occur in infective hepatitis or septicæmic states. It may be part of a reticulosis or systemic disease such as glandular fever when generalized enlargement of lymph glands is an associated finding. Large spleens are found in most patients with hæmolytic jaundice.

(*c*) Gall Bladder. Tenderness in varying degrees over the gall bladder is found in acute or chronic cholecystitis. Stones may be palpable. Gross enlargement of the gall bladder usually means that an obstruction at, or more often below, the cystic duct is present.

(*d*) Evidence of Hepatic Insufficiency. A failing liver may show certain stigmata such as the spider nævi, sallow complexion, palmar erythema, gynæcomastia, testicular atrophy and acne.

(*e*) Ascites. It is often difficult to distinguish the ascites of malignant disease from that due to cirrhosis, though the presence of intra-abdominal or pelvic masses is suggestive of malignancy. Aspiration of the fluid, which may contain demonstrable malignant cells, will make palpation easier.

(*f*) Urine. The presence of bile pigments in the urine is easily confirmed and when present virtually excludes a wholly hæmolytic process, because the renal threshold to the bilirubin of hæmolysis is only very occasionally exceeded. Neither obstructive nor hepatocellular jaundice is favoured one way or the other by the finding of bilirubinuria.

Urobilinogen is another very important pigment. Its presence in the urine in increased amounts indicates that bilirubin is being broken down by bacterial action in the intestine (very rarely by bacteria in the infected, completely obstructed bile channels), and is therefore, with this sole exception, conclusive evidence of bile pigment reaching the intestine. Its presence also indicates that the liver cell is damaged to a point of being unable to handle the normal urobilinogen turnover. It may thus appear at the beginning and at the end of a period of obstructive or hepatocellular jaundice.

In summary, urobilinogenuria is not pathognomonic of any single morbid condition, but it signifies that bile pigment has entered the intestine and functional activity of the liver cell is impaired, or rarely that there is active obstructive cholangitis. It is therefore fundamental to the appreciation of the progress of the disease in any jaundiced patient to chart daily the presence or absence of urobilinogen in the urine and its consistent absence in a jaundiced patient indicates that no bile is reaching the intestine and is much in favour of malignant biliary obstruction. In the absence of jaundice urobilinogen is found in the urine in a variety of other conditions (e.g. intestinal stasis).

(iii) Laboratory Tests

Many patients with jaundice present no great diagnostic problem and the hæmolytic cases can usually be separated without much difficulty. There will remain a few, however, of a mixed type in which a decision between an obstructive or an hepatocellular origin remains most difficult. It is in these that resort to the laboratory is required though the answer received is sometimes equivocal.

(*a*) Van den Bergh—qualitative. This test was formerly held to depend upon the existence of two distinct forms of bilirubin, bilirubin A which occurs in the serum of a typically hepatocellular jaundice and bilirubin B which is found in the presence of biliary obstruction. They give different responses to the test; a direct reaction indicating the presence of bilirubin B, and an indirect the presence of bilirubin A. Such a separation would seem positive enough were it not for the simultaneous occurrence of both types of bilirubin in just those cases which present the diagnostic difficulty. A biphasic reaction is the result.

It seems that this test is no longer held in much confidence and the older theoretical interpretations now discredited. It is doubtful whether there are two different forms of bilirubin, though its dual behaviour is not yet understood. More recently it has been stated that the erstwhile bilirubin B is not a bilirubin at all and is readily distinguished

from true bilirubin (A) by chromatographic methods and the ease with which oxidation occurs.

(*b*) Serum Proteins. The finding of a reversal of the albumen-globulin ratio in a jaundiced patient is almost diagnostic of hepatocellular disease, though long standing biliary cirrhosis may give a similar result when the liver cells are sufficiently damaged. There are other causes of a reversed ratio such as chronic nephritis, nephrosis, starvation, etc., while a hyperglobulinæmia is found in such conditions as acute infections, myelomatosis, sarcoidosis, etc.

(*c*) Thymol Turbidity. This is a flocculation test depending upon the distribution of the various protein fractions in the serum. It is the most sensitive of all the protein tests and most rapidly performed.

The normal reading lies between 0–4 units and when elevated indicates the presence of liver cell disease. A normal reading does not, however, exclude such disease. It must be emphasized that this test can only separate hepatic from extrahepatic jaundice in the early stages before obstruction has damaged the cells.

(*d*) Alkaline Phosphatase. This substance is normally excreted into the bile by the liver and in the presence of biliary obstruction of any type (except congenital atresia of bile ducts) is found in increased quantity in the serum. The normal range lies between 5–13 King Armstrong units. With very few exceptions figures of over 30 units indicate obstructive jaundice. There are at the same time other causes of a raised alkaline phosphatase as in the generalized bone destroying diseases. The phosphatase level does not necessarily parallel that of serum bilirubin and may be markedly raised in the presence of a normal bilirubin figure. Such a finding is occasionally met in primary and secondary biliary cirrhosis.

(*e*) Total Cholesterol. Apart from other diseases (such as myxœdema, nephrosis, etc.) this is raised only in obstructive jaundice, until liver damage supervenes when it may fall. A normal reading lies between 120 and 250 mgm. per 100 cc. A high reading virtually excludes hepatocellular jaundice.

(*f*) Cholesterol Esters. Normally 50–70 per cent of the total cholesterol should be esterified, but in severe liver cell damage the percentage falls and a persistently low figure carries a poor prognosis. Normal esterification is found in early obstructive lesions.

(*g*) Prothrombin. The level of circulating prothrombin may be reduced through impaired absorption of vitamin K from the acholic gut or alternatively through an inability on the part of the diseased liver cell to convert the vitamin to prothrombin. Both these mechanisms may be operative. The test when positive is of use in estimating the likelihood of hæmorrhage pre-operatively and affords a check on the effectiveness of vitamin K therapy. It is not very helpful in differentiating an obstructive from an intrahepatic form of jaundice.

There are many other means of estimating liver function. The above will give as good a guide as any. All writers agree that the best information comes from such a combination of tests rather than from any single one.

(iv) Liver Biopsy

There are many cases of jaundice (and hepatomegaly) in which all the usual liver tests are returned as normal, or their interpretation still leaves room for uncertainty.

In such a group biopsy of the liver by means of a needle gives extremely valuable information and provides a positive diagnosis in the majority of cases. It is more reliable in homogeneous rather than localized liver disease owing to the increased chances of obtaining representative material.

(*a*) TECHNIQUE. With the patient lying in bed on his back the special needle is inserted under local anæsthesia either in the ninth interspace in the mid-axillary line or subcostally. It contains a stilette which is withdrawn on reaching the liver interior. The needle is then advanced for about 3 cm. with rotation, a syringe is coupled and suction applied as the whole is withdrawn. A cylinder of liver tissue is thus obtained, though in some cirrhotics it may be difficult to secure a specimen. During the time in which the needle lies within the liver the breath is held in order to avoid unnecessary trauma. This is especially important in the intercostal route.

(*b*) CONTRAINDICATIONS. The method should not be used indiscriminately, but only in cases where the more conservative methods have failed to reach a diagnosis. The procedure carries a definite risk of hæmorrhage where there is a prothrombin deficiency or a bleeding disease or a shortage of platelets and these states should be rectified when possible before needling. Suppuration within the liver may be spread to peritoneum or pleura when an abscess exists. Hydatid livers should not be needled.

(*c*) INTERPRETATION. This is a matter for an experienced Pathologist, who bases his opinion on the following features:

(1) Cellular necrosis and its position in the lobule.
(2) Fibrosis, its age and relationship to portal tract or central vein.
(3) The type of inflammatory cell (polymorph or mononuclear).
(4) The presence of cellular regeneration and loss of the lobular architecture.
(5) The amount and distribution of bile pigments in the lobule.
(6) The multiplication of biliary channels.

In obstructive jaundice there is much pigment and increase in the number of bile channels; biliary thrombi are seen. Polymorphonuclear cells are found, especially if there is infection, and fibrous tissue accumulates around portal tracts in long standing disease.

In hepatitis there is cellular necrosis particularly at the lobule centre. Cellular regeneration and immature fibrous tissue appear in the later stages with eventual loss of architectural lobular pattern. Inflammatory cells are also seen, but polymorphs give place to mononuclears as time goes by.

Differentiation of Obstructive Jaundice. Having eliminated hepatocellular and hæmolytic jaundice it remains to attempt to distinguish between the varying causes of obstructive jaundice, which may be classified as follows:

I. Within the lumen of the bile duct:
   (1) Stone.
   (2) Inflammatory products of cholangitis.
   (3) Parasites.
   (4) Hydatid debris.

II. In the bile duct wall:
   (1) Stricture.

(2) Tumours of bile ducts. (*a*) Adenoma.
(*b*) Carcinoma.

(3) Cholangitis. (*a*) Acute.
(*b*) Chronic.
(*c*) Obliterative.

(4) Congenital deformities. (*a*) Atresia.
(*b*) Cystic dilatation.

III. Outside the wall:

(1) Pancreatic portion. (*a*) Papillary lesions: (i) carcinoma of papilla.
(ii) carcinoma of duodenum.
(*b*) Pancreatic lesions: (i) carcinoma of pancreatic head.
(ii) pancreatitis—acute, chronic.
(iii) pancreatic cysts.

(2) Retroduodenal portion. (*a*) Duodenal ulcer.
(*b*) Malignant glands, e.g. carcinoma of stomach.
(*c*) Reticulotic glands.

(3) Supraduodenal portion. (*a*) In porta hepatis, carcinomatous or reticulotic glands.
(*b*) Acute inflammations, e.g. cholecystitis.

(4) Intrahepatic portion. (*a*) Secondary carcinoma.
(*b*) Primary carcinoma.
(*c*) Gumma.
(*d*) Reticulotic deposits.
(*e*) Abscess.
(*f*) Hydatid.

IV. Primary biliary cirrhosis.

In attempting to assign the cause of obstruction in this type of jaundice it is usual first to enquire into the site of obstruction and secondly to determine whether it is due to mechanical causes or inflammation or malignancy, as judged by the length of history, age of patient, evidence of disease elsewhere, and the symptoms.

Site of Obstruction

(i) Intrahepatic

Jaundice may result from the obstruction of one or other hepatic duct. In such cases the stools may remain fully coloured despite the presence of jaundice. On the contrary it is always surprising to what extent the liver may be replaced and distorted by metastases without any jaundice at all.

(ii) Below the Gall Bladder

When the common duct is obstructed the gall bladder should always dilate unless it is either indistensible as a result of previous disease, or isolated from the increased pressure by blockage of the cystic duct, or it is absent. From this emerged Courvoisier's dictum that in a jaundiced patient the presence of a distended gall bladder favoured a diagnosis of pancreatic neoplasm rather than that of common duct stone, implying th

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  (4) Hydatid debris.

II. In the bile duct wall:
  (1) Stricture.

(c) *Completeness of Obstruction*, as judged by the prolonged and continued absence of urobilinogen in the urine, favours a malignant ætiology.

(d) *Presence of Masses* within the liver or elsewhere in the abdomen or pelvis, or of ascites may provide evidence of metastasis.

It is along these lines that this main question is often settled. The remaining causes of obstructive jaundice may be suggested by:

(i) The age of the patient, e.g. infancy and atresia of bile ducts, adolescence and cystic dilatation of common duct.

(ii) Presence of acute inflammatory signs in the right upper quadrant leading to a diagnosis of acute cholecystitis.

(iii) A history of previous gall bladder operations and the possibility of stricture formation.

(iv) Evidence of malignancy in other parts of the body.

(v) Evidence of reticulosis in spleen, lymph glands or blood count.

(vi) Residence in countries where hydatid disease or amœbiasis are indigenous.

Primary Biliary Cirrhosis

There is a form of jaundice, superficially identical with other forms of obstructive jaundice, in which at laparotomy no obstructing agent is discoverable. The ducts are not dilated and a clear extrahepatic duct system can be demonstrated. The jaundice is nevertheless obstructive and it is thought that the obstruction may be in the neighbourhood of the periphery of the lobule where the bile canaliculi join the interlobular ducts. At this point chronic inflammation and excessive fibrous tissue formation is found (pericholangiolitic inflammation), the larger bile ducts of the portal spaces being collapsed and empty in contrast to the bile duct proliferation of biliary cirrhosis secondary to extrahepatic obstruction. The fibrosis eventually invades the lobule from the periphery isolating groups of liver cells and leading at last to their destruction. Necrosis and regeneration of hepatic cells are an insignificant feature compared with the picture in portal cirrhosis.

The disease occurs for the most part in women, is insidious in onset and slowly progressive, though fluctuating. The general condition of the patient is remarkably unaffected considering the jaundice and size of the liver (which may be very large). The spleen is also palpable, and pruritis is a common early symptom.

Xanthomatosis occurs in some cases when the total serum lipoid is high for a sufficient period (over 1,800 mgm. per cent). The xanthomatous deposits are found chiefly in the skin, but may occur elsewhere such as in the arterial intima or mucous membranes. Xanthomata are more rarely seen in secondary biliary cirrhosis perhaps because the obstruction is less complete, more evanescent or more rapidly fatal.

These patients are characteristically free from pain and colic and do not have the chills of cholangitis. There is a steatorrhœa associated with the relatively acholic intestines and changes secondary to defective fat absorption such as osteoporosis and hypoprothrombinæmia.

The liver function tests are typical of an obstructive jaundice with only minor derangement of cellular function until late in the disease. The cholesterol, alkaline phosphatase, and serum bilirubin are soon elevated, while the plasma protein, thymol turbidity, etc., are little altered until the terminal stages. Finally the stage of liver failure sets in and

such a stone would have come from a diseased gall bladder. The exceptions to this good working rule are not hard to find and may be provided by instances of—

(a) *Large gall bladder with common duct stones.*

(i) Common duct stone with a mucocele of the gall bladder.

(ii) Common duct metabolic cholesterol stone and an unfibrosed gall bladder which distends.

(b) *Impalpable gall bladder with pancreatic neoplasm.*

(i) A chronically inflamed gall bladder provides no protection against neoplasm of the pancreas. The two may coexist.

(ii) The gall bladder may secure a separate opening into the duodenum.

(iii) The cystic duct may be closed by a stone, stricture or other pathology.

(iv) The gall bladder may be absent.

(v) A glandular metastasis from the pancreas may obstruct the common hepatic duct.

Chronic pancreatitis with jaundice is usually associated with chronic cholecystitis and the gall bladder seldom distends.

When pancreatic ducts are also blocked as is occasionally found when samples of duodenal juice are aspirated through a naso-gastric tube, this is a further indication of the site of the lesion.

Mode of Obstruction

(1) COMMON DUCT STONE OR PANCREATIC CARCINOMA?

This is the question most frequently posed in undoubted cases of obstructive jaundice. The answer is usually reached by consideration of the following points.

SYMPTOMS. (a) *Pain.* It is often taught that common duct stone in contradistinction to carcinoma of the pancreas is painful. While this is generally true it has been estimated that about 16 per cent of all common duct stones are painless even when causing obstruction. Pancreatic carcinoma provides an even more frequent exception to this rule and pain at some time is to be expected.

Whereas the pain fluctuates with periods of freedom in the case of stone and conforms to biliary colic, in pancreatic obstruction it is more constant, boring and wearying and more often referred to the back at a lower point than biliary colic.

(b) *Type of Obstruction.* In the same way the degree of obstruction as judged by the colour of the skin, fæces and stools is more fluctuating in the presence of stone than in malignant disease, although it is not uncommon for a patient with pancreatic carcinoma to lose his jaundice for a short time.

(c) *Duration of Disease* In some cases of common duct stone the duration of symptoms may outlast the period for which a carcinoma could have existed without destroying the patient. There may have been a long interval between attacks of jaundice.

(d) *Presence of Chills.* The occurrence of cholangitis, as indicated by chills, favours a diagnosis of common duct stone

SIGNS. (a) *State of Gall Bladder.* This has already been dealt with.

(b) *Depth of Jaundice.* The very dark pigmentation is almost only seen with malignant obstruction, though in primary biliary cirrhosis the colour is sometimes darkest of all.

not be present in the common duct. Nevertheless on rare occasions, usually associated with some chronic hæmolytic process, pigment stones may be found and in fact may form in the common duct. Either of these types of stone, probably through the medium of infection, seems to encourage the formation of biliary sludge which accretes onto the stone nucleus and at operation most stones are found to be of this clay-like consistency, with a harder centre. They often crumble when grasped with the forceps and care is needed to be sure that their removal is complete. In most cases there is a single common duct stone but it is not uncommon to find more than one, and although large numbers have been described it is rare to find more than three or four. Where multiple stones are present they are often barrel-shaped and faceted at the ends where contact with other stones is made so that an examination of such stones as are removed may provide a clue as to the existence of others.

On occasions stones are lying within the hepatic ducts at the time of operation and may, in fact, be manipulated accidentally into this situation where they are very likely to escape detection. Such a situation has been held to account for those not infrequent cases of common duct stones eventually found at laparotomy where at a previous operation a thorough negative duct exploration had been carried out. The incidence of an hepatic situation amongst common duct stones has been estimated to be about 7 per cent.

Effects of Common Duct Stones. Common duct stones earn their place of importance in surgical practice owing to the many and serious complications to which they may give rise.

Their effects may be:

(i) On the Duct

It is unusual for a stone which has been present for any length of time not to affect the lining mucosa in some way and although this may be no more than a mucosal catarrh, in more severe cases, especially when infection is superadded, actual ulceration occurs and may very rarely lead to stricture formation when the stone has gone. The presence of the stone may provide a partial obstruction to the outflow of bile and although there may have been no previous history of jaundice dilatation of the duct frequently exists. The bile stagnates and provides encouragement for the flourishing of organisms of the intestinal type. The walls of the duct become chronically inflamed, thickened, opaque, and fibrotic and the bile within cloudy, opalescent, and malodorous. In cases of severe, complete and prolonged biliary obstruction the pigment may disappear from the bile and the notorious white bile be found, which consists mainly of mucus and exudate from the wall of the duct. Furthermore the duct itself may become adherent to the neighbouring structures or the areolar tissue of the lesser omentum.

These changes are usually maximal at the lower end and diminish progressively as the ducts are ascended. At the same time it is possible, and not unusual, for stones to be present within the duct and yet none of these changes are found, so that a normal looking duct is no guarantee of the absence of a stone within.

The most severe changes are to be found where actual impaction of the stone takes place. Normally common duct stones are lying relatively free within the duct and bile is able to flow around them without much difficulty. It is only when inflammatory

hæmorrhage from portal hypertension commonly brings to an end this protracted but untreatable disease.

Surgically it is important in so far as it is virtually inseparable from other causes of obstructive jaundice, though the above mentioned features may serve to eliminate some of the possible causes of obstruction. *Such cases will and should come to laparotomy* when the condition may be suspected in the presence of a large mildly cirrhotic liver and normal biliary passages. A liver biopsy will give the most solid basis for such a diagnosis, though it should not be used as an argument against surgical exploration. Confronted with these operative findings the surgeon is at a loss to find an explanation and it is tempting sometimes to assume that a common duct stone has passed on into the duodenum just prior to operation. This may sometimes happen but again the biopsy should distinguish between primary and secondary biliary cirrhosis by the condition of the ducts in the portal spaces.

This disease is not excessively rare and its ætiology is unknown.

References

(1) Ahrens, E. H., Jr. et al. (1950) *Medicine*, Baltimore. 29, 299.
(2) Cantarow, A. and Trumper, M. (1946) Clinical Biochemistry, 3rd. Ed., *W. B. Saunders & Co.*, Philadelphia.
(3) Cole, P. G. and Lathe, G. H. (1953) *J. clin. Path.* 6, 99.
(4) Lichtman, S. S. (1953) Diseases of the Liver, Gall Bladder and Bile Ducts, 3rd Ed., *Henry Kimpton*, Philadelphia and London.
(5) MacMahon, H. E. and Thannhauser, S. J. (1949) *Ann. intern. Med.* 30, 121.
(6) Thannhauser, S. J. (1950) Lipidoses, diseases of the cellular lipid metabolism, 2nd Ed., *Oxford University Press.*

COMMON DUCT STONES

Incidence. It is almost certain that gall stones find access to the common duct very much more frequently than any of the published figures indicate and no doubt the majority of these pass safely into the duodenum, causing no more than transient symptoms. It is only when a stone of sufficient size passes through the wide cystic duct but fails to negotiate the duodenal papilla that trouble is likely to ensue. A stone which rests within the common duct for any length of time can easily grow in size or accumulate a coating of biliary mud. Although the majority of common duct stones originate in the gall bladder it is generally believed that stone formation can occur in cases where the gall bladder has previously been removed. There is always some element of doubt, however, as it is never possible to say certainly that a small calculus, perhaps within the hepatic ducts, may not have been overlooked at the time of cholecystectomy.

The likelihood of coexistent common duct stones at the time of cholecystectomy has been variously assessed at about one in four. This figure is of great importance as an indication for the more frequent exploration of the common duct at the time of cholecystectomy.

Type of Stone. Most common duct stones are to be found in patients whose gall bladder has been diseased and it follows that these will be mixed stones in most cases, though there may be a cholesterol nucleus. Their previous origin in the gall bladder may often be recognized by the multiple facets from the proximity of other stones which may

not be present in the common duct. Nevertheless on rare occasions, usually associated with some chronic hæmolytic process, pigment stones may be found and in fact may form in the common duct. Either of these types of stone, probably through the medium of infection, seems to encourage the formation of biliary sludge which accretes onto the stone nucleus and at operation most stones are found to be of this clay-like consistency, with a harder centre. They often crumble when grasped with the forceps and care is needed to be sure that their removal is complete. In most cases there is a single common duct stone but it is not uncommon to find more than one, and although large numbers have been described it is rare to find more than three or four. Where multiple stones are present they are often barrel-shaped and faceted at the ends where contact with other stones is made so that an examination of such stones as are removed may provide a clue as to the existence of others.

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The features of chronic cholecystitis have already been dealt with and are or have been present in most patients who eventually develop common duct stones. Indeed such a stone may produce the typical symptoms of cholecystitis though the gall bladder has already been removed.

(i) COLIC

There is no doubt that stones can exist in the common duct without causing any pain whatsoever and some doubt has been cast as to whether true biliary colic ever results from a stone in this situation, especially as the amount of smooth muscle in the common duct is variable and usually very small. However, difficulties arise from the high incidence of associated stones in the gall bladder and the fact that most common duct stones have at one time or another negotiated the cystic duct. From a practical point of view many patients have suffered from a biliary colic-like pain and whether this was coming from the gall bladder or the common duct is never certainly known. It is practically never possible to decide from the type or distribution of the pain whether a stone in the common duct or gall bladder is causing it.

Furthermore, post-cholecystectomy pains of varying type are not uncommon and from the character of such pains it can seldom be said whether they result from an overlooked stone or some functional disturbances of the remaining biliary tract.

The distribution of pain due to a common duct stone need not differ greatly from that already described due to stones within the gall bladder. It is perhaps more frequently epigastric and felt anteriorly.

(ii) JAUNDICE

This is not necessarily present at the time of examination but is likely to have occurred or may occur in the future in most cases. A characteristic feature is that it is intermittent and variable and that its onset tends to coincide with the other symptoms, such as pain and vomiting.

The jaundice is obstructive in type and during attacks of sufficient duration is associated with choluria and a pale stool. It must be remembered, however, that as the obstruction is usually incomplete and short-lived it may be possible for normal stools to be passed throughout the attack. In patients who are not clinically jaundiced the urine must be tested for the presence of bile as such a finding, though often fleeting, is of the utmost diagnostic value in a difficult case.

(iii) CHILLS

The acute episodes in the symptomatology of common duct stones are usually precipitated by a transient infective process within the common duct which makes the partial obstruction more complete by the swelling of the duct wall and by the accumulation of mucopus and debris within the duct. Such infective episodes are often characterized by the "intermittent hepatic fever" of Charcot. These attacks consist of sharp bouts of fever, in severe cases reaching 105°F, associated with severe systemic upset. The patient suffers minor chills in mild cases or actual rigors when severe, and feels extremely ill. There is often profound nausea, vomiting, sweating, and prostration. Such an attack probably indicates that organisms have begun to flourish throughout the biliary tree and that owing to the tension of the obstructed bile considerable

swelling of the duct wall or the impaction of a stone make the obstruction more complete that jaundice ensues and the pathology becomes more acute. The process is notoriously intermittent.

(ii) On the Liver

The liver is adversely affected more frequently than is realized and in the presence of recurrent attacks of hepatic fever and jaundice it is not surprising to find the changes of biliary cirrhosis, but in those cases where stones are present and in which jaundice has not been noticed hepatic damage is much more frequent than would have been expected and wherever the possibilities of common duct stone exist a pre-operative estimation of hepatic function may warn the surgeon of an additional risk and possible post-operative complications. It thus seems possible that obstruction and biliary infection may exist over quite long periods of time without necessarily giving rise to clinical jaundice or fever of a sufficient degree to attract the patient's attention and yet produce definite histological changes in the liver parenchyma. These consist of cellular atrophy, hypertrophy of the biliary canaliculi and the formation of biliary thrombi, fibrous tissue, and chronic inflammatory cells. When these changes reach a severe degree fatty atrophy and necrosis around the portal tracts are also found. In the presence of jaundice the liver substance is stained with bile pigment.

In other cases the suppurative factor is more pronounced and acute inflammatory cells are to be found congregated around the finer bile channels and in places definite pus formation of a widespread focal distribution can be demonstrated. Such findings constitute suppurative cholangitis and multiple cholangitic liver abscess formation.

All these changes in greater or lesser degree hamper normal processes of liver function and increase the operative risk in the individual case. Their effects can be diminished by careful pre-operative preparation with the administration of antibiotics, vitamin K, glucose together with electrolyte, hæmoglobin, and perhaps protein restitution. More important, however, than all these is the early establishment of free biliary drainage which may be required as a preliminary to more extensive intra-abdominal procedures.

(iii) Pancreas

The effects of common duct stone upon the pancreas have previously been mentioned and the association between such pancreatic pathology and gall stones is well known. The presence of stones within the duct could conceivably affect pancreatic function by:

(1) Pressure on the pancreatic duct

(2) Inflammation spreading from the common duct to the pancreatic duct.

(3) Reflux of bile into the pancreatic duct where anatomical arrangements favour such an occurrence

(4) Partial or complete obstruction of the pancreatic ducts themselves due to a stone impacted in the ampulla

Symptoms. It is important to realize that common duct stones may be entirely symptomless or masked by the symptoms of the associated cholecystitis. Common duct stones are found at postmortem in patients who have had no indication of their presence during life. It is estimated that approximately 25 per cent of all common duct stones have never caused jaundice although the chances of their doing so increase with the length of time during which they are permitted to remain.

itself is often palpably distended. The history is short by comparison with cases of stones.

Diagnosis. Before cholecystectomy and in the presence of gall bladder disease jaundice and chills alone provide evidence of common duct stones. The differential diagnosis of jaundice is considered in the chapter on this subject.

After cholecystectomy persistence of symptoms or their recurrence may be due to:

(1) A mistake in the original diagnosis.
(2) A persisting common duct stone.
(3) A stricture of the bile ducts.
(4) Pancreatitis.
(5) Biliary dyskinesia, including spasm or fibrosis of the sphincter of Oddi.
(6) A persistent diseased gall bladder stump.
(7) Post-cholecystectomy syndrome.

These states are discussed under "results of cholecystectomy."

When the above-mentioned features are present the diagnosis may be easy, but when dyspepsia and chronic pain without chills or jaundice provide the only clues there is great difficulty. The use of choledochography and a repeated search for bile in the urine during attacks provide the main hope of proving the presence of a stone. A stricture may give identical symptoms. In severe cases laparotomy may be indicated as the only means of excluding a remediable cause of suffering.

Investigation by X-rays

(1) Cholecystography may by revealing stones in a diseased gall bladder expose the likelihood of a common duct stone which may itself be visible on straight films overlying the right transverse process of the upper lumbar vertebræ.

(2) The more recent introduction of Biligrafin, a substance given intravenously, may revolutionize the diagnosis of biliary duct pathology in that it is not dependent on the presence of the concentrating power of the gall bladder in order to be made visible. In this way the actual common duct itself may be outlined and filling defects noted even after cholecystectomy (Fig. 23). Further experience is required with the method before its shortcomings are fully known

Treatment. The pre-operative treatment does not materially differ from that described for cholecystectomy in the presence of jaundice. The important aspects are concerned with the use of preparatory antibiotics when infection is present, the administration of vitamin K and glucose in the presence of jaundice and possible liver damage and the selection of the most favourable time for operation. In past years surgeons would often observe jaundiced patients believed to be suffering from common duct stone for weeks on end in the hope of arriving at a time when the jaundice would relent and the risks of surgery accordingly diminish. This was before the use of vitamin K and antibiotics which have largely freed the surgeon from such a waiting policy. Nevertheless, there is seldom a binding urgency for operation and a few days employed in improving the patient's condition is well worth while, when such methods appear to be having some effect.

absorption of their products into the systemic circulation is taking place. Such a multiplication of organisms does not necessarily imply a suppurative process within the duct wall.

The chills and rigors usually coincide with the onset of other symptoms of common duct stone. They are seldom seen in cases of obstructive jaundice without infection of the biliary tree. It is this very association of the above-mentioned symptoms, at intervals, *which forms the strongest point in the diagnosis of common duct stones* and of all symptoms that of jaundice is of most use in separating the patients with stone from those with functional biliary dyspepsia and pain.

The clinical picture varies according to whether the stone is uncomplicated or associated with other pathological changes. The following types of syndrome may be presented:

(1) The stone may be completely silent.
(2) Chronic dyspepsia.
(3) Biliary colic with or without obstructive jaundice.
(4) Intermittent obstructive jaundice with or without pain.
(5) Unrelenting obstructive jaundice.
(6) Jaundice with cholangitis or even liver abscess.
(7) Chronic or subacute hepatic failure.
(8) Portal hypertension.
(9) Post-cholecystectomy "dyschinesia."
(10) Acute or chronic pancreatitis.

The groups are by no means watertight and frequently merge one with another. In all such cases suspicion is centred upon the possibility of a common duct stone.

Following cholecystectomy the persistence or recurrence of symptoms often similar to those preceding operation immediately opens up the possibilty of common duct stone.

Physical Signs. The physical signs are very often absent but may include a large liver which is tender or hard. The urine contains bile salts, pigments and urobilinogen. The skin and scleræ may be stained and the stools pale, though the degree of obstruction varies from day to day. Hepatic insufficiency may lead to spider nævi, palmar erythema, gynæcomastia, ascites, etc. The spleen is occasionally palpable and enlarged abdominal veins are visible when cirrhosis is severe. The scar of a previous cholecystectomy is often present. Such findings are only present in severe long standing cases and bear evidence to the complication rather than to the stone itself.

In the presence of obstructive jaundice due to stone the gall bladder is characteristically not enlarged (Courvoisier's law) as it is usually fibrotic, small and indiscernible. This contrasts with jaundice due to pancreatic lesions or extra biliary pressure. The presence of an associated mucocœle of the gall bladder provides an exception to the rule. The patient may run an intermittent fever with a daily rise to 100°F; or rigors with pyrexia up to 105° may be a feature in severe cases.

By contrast with these findings the jaundice due to pancreatic carcinoma is characteristically complete and unremitting. The stools are consistently pale and there is no urobilinogen. Fever is absent and the liver is more enlarged, while the gall bladder

is then available for examination. If it is turbid or particularly black and viscid the duct should be explored.

(5) The state of the tissues around the lower end of the bile duct.

When on palpation of the lower end of the bile duct and head of pancreas the tissues are thickened and there is loss of definition between the various structures or suspicious hard areas are felt the duct should be probed to exclude the presence of stones.

(6) When undoubted biliary symptoms cannot be explained by the condition of the gall bladder alone.

Whereas the above indications are useful guides, in the experience of most surgeons there are well-remembered cases in which common duct stones were present and yet none of the above indications existed. It is consequently a difficult matter in the absence of such pointers to know when to explore, and sometimes stones are found when the duct is opened almost by instinct.

There has been a great increase in the frequency of choledochotomy at the time of cholecystectomy during recent years and some clinics explore as many as 50 per cent of their cases and most experienced surgeons do so in at least 25 per cent. It is true to say that the incidence of common duct stones increases with the frequency of choledochotomy. If the true incidence of common duct stones is in the region of 25 per cent of all cases coming to cholecystectomy, then a figure greater than this should represent the proportion requiring choledochotomy. There is very little disability associated with choledochotomy and no guilt should be attached to a negative exploration when no stones exist. In fact the subsequent drainage of an abnormal common duct may be beneficial despite the absence of stones.

The practical details are fully described in the chapter upon operative surgery.

If no stones are found but a close examination cannot establish a completely clear channel to the duodenum, or the pancreas is not above suspicion, it is as well to reflect before removing it that the gall bladder may prove useful as a means of by-passing at a later date if not already too diseased. It is not rare for a surgeon in difficulties with low biliary obstruction to regret the earlier removal of the gall bladder by himself or a colleague.

(ii) Late Operation

When the gall bladder has previously been removed the primary object of the operation is the exploration of the common duct and the surgeon need not worry himself with operative indications for choledochotomy.

Operation is indicated when, after cholecystectomy:

(1) Undoubted attacks of obstructive jaundice ensue.

(2) Chills and rigors, often associated with jaundice, are recurrent.

(3) Relapsing pancreatitis demands the exclusion of a common duct stone.

(4) A filling defect is visible on choledochography.

(5) Pain, dyspepsia and vomiting are particularly severe and seem to be originating in the biliary tract. It is only after a negative exploration that a concrete diagnosis of dyskinesia becomes tenable.

The methods do not differ materially from those employed during choledochotomy at the time of cholecystectomy.

(i) At the Time of Cholecystectomy

The time for treatment of common duct stones is during the first cholecystectomy. It is at this stage that subsequent ill health, disappointments and secondary operation can be avoided. It is consequently of considerable importance to know when the common duct should be explored. At all times during any operation in the biliary region thorough

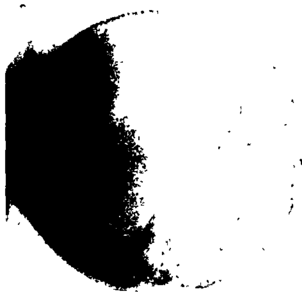


Fig. 71 Intravenous choledochogram using Biligrafin and showing a large calculus at the lower end of the common duct

palpation of the common duct must be carried out, but the duct itself is only explored *when certain indications are present.*

These are:

(1) A history of jaundice.

(2) The anatomical arrangements of the biliary ducts.

When the gall bladder contains small stones, particularly when it is contracted or when the cystic duct is of large calibre relative to the stones it is highly probable that stones also exist within the common duct.

(3) Alterations in the common duct.

Dilatation or thickening of the duct wall are positive indications for exploration. It should be remembered, however, that the common ducts are nearly always dilated following previous cholecystectomy secondary to the loss of the reservoir and safety valve function of the gall bladder. In the same way, when the gall bladder is eliminated by disease such a dilatation may also occur. This can explain certain cases of dilatation in which no stone is found, but such an explanation should not be used as an excuse for not exploring the duct.

(4) The state of the bile.

It is a good practice to aspirate the common bile duct as a matter of routine for not only does this distinguish the bile duct from the portal vein, but also a specimen of bile

the duct system is estimated by a further picture about 10 minutes after the first. A water-soluble dye is for this purpose much more satisfactory than an oily one because of its miscibility with the bile and the absence of globule formation. Care should be taken not to introduce air bubbles as these may lead to filling defects indistinguishable from those caused by a stone (Fig. 72).

The T-tube is normally retained for a period of 12–16 days when convalescence goes to plan. When infection and dilatation persists or cholangiography reveals suspicious areas within the duct the T-tube is retained until such time as these unsatisfactory features disappear, sometimes for two or three months. Where it seems probable that a stone is still retained within the duct a further operation may be indicated, but prior to this certain conservative measures have been advocated as a method of dealing with such stones when a T-tube is *in situ*. Various solutions have been introduced, such as ether or chloroform, with the intention of dissolving or breaking up the stone. These methods can be combined with attempts to secure relaxation of the sphincter of Oddi by means of nitrites, fatty food, etc., while the flow of bile may be increased by the use of chologogues such as bile acids. These methods are not often availing and stones subjected to the ether treatment *in vitro* showed remarkable resistance to these measures, though no doubt the physical effect of lavage is more useful than the dissolving properties of the solutions. Nevertheless, successful cases are on record, and happy the surgeon and patient who is thereby exonerated from further operation.

There are yet a few patients in a very poor state, either as a result of intense jaundice or severe cholangitis or suffering from acute pancreatitis in whom the handling necessary for a complete exploration of the common duct seems ill-advised. In these cases it is sometimes expedient to be satisfied with the drainage of the biliary tree above the site of the obstruction. This can be done by cholecystostomy when it is certain that such a manœuvre provides an unobstructed drainage, or perhaps better by a T-tube inserted in the supraduodenal portion of the duct. In this way the patient's condition may improve sufficiently to allow secondary operation at a time of choosing, when the risks are considerably less.

References

(1) Allen, A. W and Wallace, R. H. (1940) *Ann. Surg.* **111**, 838.
(2) Corff, M., Bergers and Gershon-Cohen, J. (1952) *Surg. Gynec. Obstet.* **94**, 294.
(3) Glenn, F. (1952) *Surg. Gynec. Obstet.* **95**, 431.
(4) Judd, E. S. and Marshall, J. M. (1930) *J. Amer. med. Ass.* **95**, 1061.
(5) Lahey, F. H. (1937) *Ann. Surg.* **105**, 763.
(6) Lahey, F. H. (1945) *Surg. Gynec. Obstet.* **80**, 197.
(7) Mayo, W. J. (1923) *Lancet*, **205**, (i), 1299.
(8) Pratt, G. F., Saner, W. G. and Gray, H. K. (1951) *Gastroenterology*. **18**, 443.
(9) Snell, A. M. (1936) *Surg. Gynec. Obstet.* **63**, 596.
(10) Trueman, K. R. (1940) *Proc. Mayo Clin.* **15**, 283.
(11) Walters, W. and Snell, A. M. (1940) Diseases of the Gall Bladder and Bile Ducts. *W. B. Saunders & Co.*, Philadelphia.
(12) Walton, J. (1940) *Brit. J. Surg.* **27**, 295.
(13) Watson, C. J. (1947) *Gastroenterology*. **9**, 752.

It may not always prove possible to remove a stone which is impacted in the lower end of the common duct through a supraduodenal choledochotomy. In such cases the stone may be approached by a transduodenal route and the papilla directly visualized within the lumen of the duodenum. At such a time the papilla itself may be incised (McBurney), or an incision made in the posterior duodenal wall overlying a stone impacted some distance above the papilla (Kocher).

At other times a retroduodenal or retropancreatic approach is made and the duct exposed by the mobilization, retraction and medial dislocation of the duodenal crook and pancreas, and indeed this manœuvre is often useful to facilitate a more thorough palpation of the duct.



Fig. 72 A post-operative cholangiogram, through a T-tube, showing what every surgeon hopes never to [illegible]

The common duct is invariably drained following choledochotomy. This is because following manipulation of this region there may be some impediment, as a result of œdema or spasm, of the outflow of bile into the duodenum during the immediate post-operative period. This would cause a pressure within the duct and lead to a leakage through the incision in the common duct and consequent biliary peritonitis. This risk is almost entirely avoided by the insertion of a T-tube. Furthermore, in some cases free post-operative biliary drainage is a very important factor in the treatment of the associated cholangitis and in the recuperation of the damaged liver. Such a freedom of drainage cannot be guaranteed in any other way. Many of the fatalities of operations upon the biliary tree in bad-risk patients with jaundice and cholangitis were due to an exacerbation of the obstruction and inflammation as a result of surgical manipulation. Free drainage circumvents this immediate post-operative risk. A T-tube provides valuable information during the post-operative period of the progress of events within the duct. The issuing bile may be inspected for turbidity and cultured so that the persistence of infection may be recognized and bacterial antibiotic sensitivity tested. The quantity of bile discharged from the tube is equal to the quantity secreted by the liver minus the amount which has passed into the duodenum. In this way a fair estimate of the patency of the lower end of the duct can be made and the effects of closing the issuing limb of the tube observed. Where obstruction at the lower duct end persists closure of the tube results in pain or the discharge of bile around the tube or even recurrence of the jaundice.

At a later date cholangiography should be carried out as a routine in order to visualize the entire duct system Any filling defects may indicate the presence of a stone which has been overlooked, or persistent dilatation may suggest that obstruction remains. The ease with which the dye reaches the duodenum and the rate of clearance of the dye from

the duct system is estimated by a further picture about 10 minutes after the first. A water-soluble dye is for this purpose much more satisfactory than an oily one because of its miscibility with the bile and the absence of globule formation. Care should be taken not to introduce air bubbles as these may lead to filling defects indistinguishable from those caused by a stone (Fig. 72).

The T-tube is normally retained for a period of 12–16 days when convalescence goes to plan. When infection and dilatation persists or cholangiography reveals suspicious areas within the duct the T-tube is retained until such time as these unsatisfactory features disappear, sometimes for two or three months. Where it seems probable that a stone is still retained within the duct a further operation may be indicated, but prior to this certain conservative measures have been advocated as a method of dealing with such stones when a T-tube is *in situ*. Various solutions have been introduced, such as ether or chloroform, with the intention of dissolving or breaking up the stone. These methods can be combined with attempts to secure relaxation of the sphincter of Oddi by means of nitrites, fatty food, etc., while the flow of bile may be increased by the use of chologogues such as bile acids. These methods are not often availing and stones subjected to the ether treatment *in vitro* showed remarkable resistance to these measures, though no doubt the physical effect of lavage is more useful than the dissolving properties of the solutions. Nevertheless, successful cases are on record, and happy the surgeon and patient who is thereby exonerated from further operation.

There are yet a few patients in a very poor state, either as a result of intense jaundice or severe cholangitis or suffering from acute pancreatitis in whom the handling necessary for a complete exploration of the common duct seems ill-advised. In these cases it is sometimes expedient to be satisfied with the drainage of the biliary tree above the site of the obstruction. This can be done by cholecystostomy when it is certain that such a manœuvre provides an unobstructed drainage, or perhaps better by a T-tube inserted in the supraduodenal portion of the duct. In this way the patient's condition may improve sufficiently to allow secondary operation at a time of choosing, when the risks are considerably less.

References

(1) Allen, A. W. and Wallace, R. H. (1940) *Ann. Surg.* 111, 838.
(2) Corff, M., Bergers and Gershon-Cohen, J. (1952) *Surg. Gynec. Obstet.* 94, 294.
(3) [illegible]

[illegible] 443.

(10) [illegible]
(11) [illegible] & Co., Philadelphia.
(12) Walton, J. (1940) *Brit. J. Surg.* 27, 295.
(13) Watson, C. J. (1947) *Gastroenterology*. 9, 752.

STRICTURES OF THE BILE DUCTS

THE surgeon is called upon to repair the bile ducts when these have been damaged by trauma, infection or new growth or when through a failure of development they are atretic.

These cases are not numerous but are particularly cogent when they do occur. Apart from the congenital malformations these unfortunate people are frequently victims of previous surgery to which in many cases they may owe their disability. Their disease is a particularly distressing one in so far as they are not old, the lesion is so small and not in itself lethal or necessarily progressive: but its effects are terrible. The jaundice cannot be hidden and prevents normal social intercourse; the itching interferes with sleep, a biliary fistula is messy and exhausting and the frequent chills a recurring source of malaise and misery. The liver damage is progressive; appetite is destroyed and digestive trouble almost constant until life is scarcely worth while. On top of this it is usual for the patient to have suffered the hopes and disappointments of more than one operative attempt to restore internal biliary drainage.

There seems no doubt that this disease is much more prevalent in America as can be judged from the prominence given to this topic in their surgical literature. This may be due to the higher incidence of gall bladder disease and also to the vast areas far removed from the larger surgical centres in which acute cholecystitis must be dealt with where it occurs, under conditions that are not ideal. In these circumstances accidents are prone to occur. In this country strictures of the bile ducts are not common though it has been stated that they may become more so with the increasing decentralization of surgery

Ætiology. Benign strictures of the bile ducts are due in most cases to injury of the ducts during previous operations; these are usually cholecystectomy and a very few following gastrectomy for duodenal ulcer. Various authors have assessed the proportion of post-operative cases at about 80 per cent of the total.

Causes of benign stricture of the bile ducts:

(1) Trauma.

(*a*) Operative	cholecystectomy
	gastrectomy.
(*b*) External violence .	penetrating.
	non-penetrating.

(2) Stones in common bile duct.
(3) Obliterative cholangitis.
(4) Peptic ulcer of duodenum
(5) Pancreatitis.

TRAUMA

Those occurring during cholecystectomy are for the most part avoidable and due to insufficient care in identifying and handling the various structures.

Damage can be avoided by ensuring the following safeguards:

(1) An adequate incision in a relaxed patient.
(2) Packing away the unwanted viscera with full exposure of the porta hepatis.
(3) Efficient lighting.

(4) A knowledge of the known variations in anatomy, not only of the bile ducts but also of the vessels. Each patient should be approached as though a new species was being dissected for the first time as no amount of foreknowledge can foretell the anatomy in the individual patient.

(5) No structure should be divided until hepatic, cystic, and common bile ducts, the right hepatic and cystic arteries are clearly demonstrated to the satisfaction of an assis-



FIG 73 Danger! Blind and inaccurate application of artery forceps Injury is done to hepatic arteries and the common bile duct, resulting in "liver deaths" or late stricture

tant, and even then clamps are better avoided. All ligatures are passed precisely with aneurysm needles.

(6) In the event of hæmorrhage bleeding may be arrested with safety by manual compression of the free edge of the lesser omentum with a finger in the foramen of Winslow. Meanwhile the field is sucked clean and the bleeding point identified and secured by momentary relaxation of the pressure. In this way blind clamping is avoided, and danger averted.

(7) An inspection of the specimen removed and the structures remaining at the end of the operation may enable any mistakes to be rectified at once; such an opportunity will not come again.

Errors are most likely to occur in fat persons insufficiently relaxed, when the surgeon, with poor assistance, is struggling against time at the bottom of a deep, dark, bloody hole. A clamp is thrust blind in the direction of the source of hæmorrhage and the bile ducts are accidentally included (Fig. 73)—this results in a crush and subsequent necrosis

followed by fibrosis or perhaps even a post-operative fistula; or, the bleeding point secured, a ligature includes part of the duct producing either a complete or partial obstruction.

Another danger occurs when traction on the cystic duct so angulates the common duct as to drag it into the shape of a Y. In this position the true point of junction cannot accurately be judged and a clamp and subsequent ligature may bite off part of the

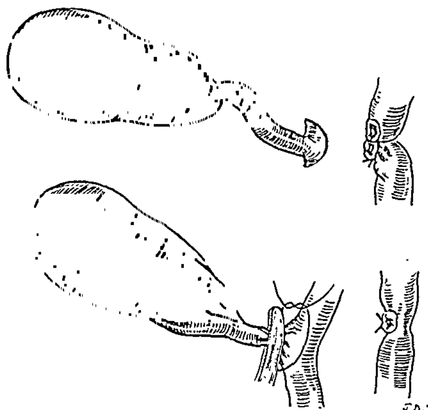


FIG 74 Classical mistakes: (i) An "easy" cholecystectomy. Too much traction on the cystic duct has led to excision of part of the common duct. (ii) In ligating the cystic duct the artery forcep is too close to its origin, resulting in a narrowing of the common duct lumen The use of aneurysm needles is preferred for the placing of ligatures in this area

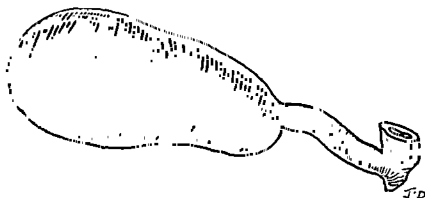


FIG. 75. The cholecystectomy specimen should always be inspected for such surprises The damage is more easily repaired immediately after it is done

circumference of the main ducts. This accident is more likely when the cystic duct is missing and the gall bladder opens direct into the common duct (Fig. 74).

On occasions a complete segment of the common duct may be excised along with the gall bladder. This error is recognized if the specimen is inspected as a routine after removal (Fig. 75).

The demonstration of anatomical feature is most difficult when a stone impacted in Hartmann's pouch has caused much surrounding inflammation which involves or even

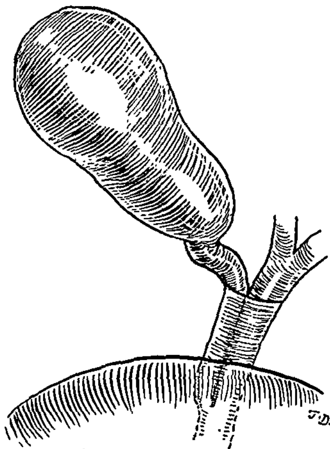


FIG. 76. A low entry of the cystic duct after sharing a common sheath with the hepatic duct.

ulcerates into the adjacent hepatic duct. In these cases the medial hepatic duct wall is mistaken for the wall of the gall bladder and leads to excision of the common hepatic duct.

Where the cystic duct is long and closely bound to an equally long common hepatic duct there is also danger of excising the combined structure (Fig. 76).

Narrowing of the common duct may result from an over meticulous closure of the free border of the lesser omentum. Abscess or granuloma formation in this area as a result of biliary leakage from an unrecognized accessory duct or other site, may cause late fibrosis.

During a search of the ducts with probes or while dilating the ampulla sufficient damage may be done to cause later constriction.

During gastrectomy the retroduodenal portion of the duct may be injured where a

penetrating posterior wall ulcer is situated some distance beyond the pyloric ring. If the neighbourhood of the duct is reached while freeing the duodenum it is as well to insert a T-tube through the duct in order to be sure of its whereabouts, or alternatively to be content with excluding rather than resecting such an ulcer where vagotomy is not indicated.

These are the classical errors which cannot be too well known if tragedies are to be avoided.

Infection

The factor of infection both within and outside the ducts is hard to assess but that narrowing can arise independently of any injury is known from those cases of obliterative cholangitis involving intrahepatic (as well as extrahepatic) channels where no surgeon could ever have ventured an erring instrument. In these cases the lining mucosa is sometimes missing. Organisms are nearly always present and often active in the common ducts at the time of cholecystectomy and their control by antibiotics or chemotherapy lessens a possible source of trouble. It is suggested that at least on some occasions the gangrenous inflammation which is involving the gall bladder during an acute cholecystitis may extend to the ducts themselves causing later fibrosis.

The other causes of stricture, approximately 20 per cent of the whole, arise insidiously and not much can be done to prevent their onset until more is known about their ætiology.

The Effects of Stricture of the Bile Ducts

These will largely depend on whether the obstruction is partial or complete, and with or without external biliary fistula. Many cases vacillate between these states—the fistula heals from time to time only to produce another attack of obstructive jaundice followed by recurrence of the fistula either externally or internally. A fistula is never static and is the resultant of a constant antagonism between the natural power of healing and the pressure of infected bile seeking exit. Free drainage is never really established.

The Ducts. The ducts above the obstruction dilate well into the liver substance and become acutely inflamed during the episodes of infection, which are frequent.

The Liver. These attacks of infection may at any time culminate in multiple cholangitic abscesses with perhaps a fatal conclusion and in any event leave the liver more crippled than previously. Fibrosis of liver substance accompanies the healing during the quiescent periods and eventually leads to a full blown biliary cirrhosis of the Hanot type. Congestive splenomegaly due to portal hypertension is the sequel in longstanding cases and gastro intestinal hæmorrhage may finish the story.

Apart from suppuration and fibrosis the individual liver cell suffers and can frequently be shown by the crude liver tests available to be reduced in efficiency. Such a deterioration usually accompanies the cirrhotic changes, but is not necessarily proportional to them and may be demonstrable in the absence of macroscopic cirrhosis.

Deficiency States When a biliary fistula is present for any appreciable length of time it has two main effects upon the individual. There is firstly a loss to the surface of basic ions and chlorides and of bile salts which must all be replaced if equilibrium is to be maintained.

Secondly, the lack of bile in the intestines leads to steatorrhœa and absorptive difficulties, especially with the fat soluble vitamins D and K and calcium, resulting in

reduced blood prothrombin levels and osteoporosis. The resulting hæmorrhagic state may show as a purpura and was responsible for the high post-operative mortality from hæmorrhage in other times.

Clinical Picture. In those cases where injury is done to the bile ducts at operation symptoms may be immediate or delayed.

The immediate results are seen during the first post-operative days. If the ducts are completely obstructed jaundice appears and deepens progressively. The duct may give way where a stitch encloses it and result in a flooding of bile either into the peritoneal cavity or through the wound if a drain remains, and a fistula is established. This is usually permanent where no passage leads to the duodenum though occasionally a spontaneous internal fistula develops after stenosis of the external sinus.

If the duct has been crushed there may be no immediate leakage, but over a course of months a progressive fibrosis gradually constricts it; or the lumen may have been actually narrowed at the original operation. This state may give rise to no symptoms for a period of years but is comparable to a urethral stricture with stagnation above it. Chronic infection sooner or later becomes irrevocably established and deposition of sludge serves only to increase the inadequacy of the drainage. On top of this an acute inflammatory flare-up in the duct wall from time to time completes the obstruction so that the jaundice is intermittent and may strongly suggest a common duct stone.

Any discharge of bile post-operatively which does not cease within the first week should strongly suggest residual obstruction or injury to the bile ducts, as does obstructive jaundice dating from the immediate post-operative days; though an overlooked common duct stone may also produce these effects.

The late jaundice or stricture will not usually be differentiated from that of a common duct stone and the true ætiology is often only recognized at operation. Chronic pancreatitis, recurrent cholangiohepatitis and carcinoma of the pancreas may simulate both.

Rigors, chills, and pyrexia of the Charcot type betoken the cholangitic attacks and usually herald a reappearance or deepening of pre-existing jaundice. Pain of a constant nature is experienced during exacerbations only, while vomiting and dyspepsia are usual accompaniments. Pruritis may be the most distressing part of the syndrome.

The history of a previous cholecystectomy which was performed with difficulty and followed by a stormy convalescence is strongly suggestive of stricture in a patient who later becomes jaundiced. There are also a few patients some of whom have undergone previous cholecystectomy who now suffer recurrent jaundice attacks and in whom subsequent exploration reveals no stone or stricture to account for it. On the one hand, in this group are those suffering a recurring infective cholangitis without apparent reason, and on the other, the cases of cholangiolytic hepatitis. In each treatment is most difficult. Another group will be found to be due to recurrent virus hepatitis presumably unconnected with previous surgery. In each type some cirrhosis is not uncommon. A previous liver biopsy might serve to separate these from the purely obstructive cases.

Treatment. These conditions are notoriously difficult to treat. The surgeon starts with a discouraged patient whose faith in surgery is already severely shaken. In many instances previous attempts at repair may have been made on more than one occasion and each unsuccessful attempt is almost bound to have left the patient with less duct

than he originally had. There is usually emaciation and some degree of liver damage. The operation is never easy and the results by no means certain.

Repairs of the ducts are required in two circumstances—either immediately after injury or at a later time. The ideal moment to repair is at the time of the damage for then it is easy and the chances of success are at their best: the extent of the injury is limited and secondary changes in duct, liver, and patient are not yet established. It is for this reason that a careful inspection of all structures should be carried out before closing the abdomen. If a duct has been incised it should be accurately resutured and T-tube drainage provided to remove all pressure during the healing phase. A crushed area should be excised, not necessarily by removal of the complete circumference of the duct as a small wedge can be removed from the side and adequate closure is still possible. Too much stripping of the ducts should at all times be avoided as there may be some danger of depriving them of their arterial supply.

It is in the *later phases*, with the initial injury passed unrecognized, that the ingenuity and patience of the surgeon is fully taxed. The problem is a difficult one which has intrigued and challenged some of the best brains and hands in surgery.

The difficulties may be summarized as those of:

(1) Approach and isolation of the duct ends.

(2) The mechanical joining of the ducts where possible.

(3) Preventing further stenosis.

(4) Compensating for the absence of the sphincter of Oddi where the duct must be joined to intestine.

(5) Securing a live patient at the end.

The old conservative attitude to obstructive jaundice has changed in recent years. Surgical interference was associated with so high a mortality, chiefly from hæmorrhage, as to persuade our predecessors to watch their jaundiced patients for months at a time hoping for a remission. Our changed attitude is due to increased knowledge of the bleeding fault and our new powers to put it right. The present trend is towards active and early surgical correction of the obstruction.

Pre-operative Treatment

Owing to the poor state of these patients their general condition must receive much attention before any major surgery is attempted.

Patients with a fistula of any magnitude must receive a complete electrolytic investigation. They are likely to be wanting both sodium and chloride in addition to being dehydrated and acidotic with decreased CO_2 combining power of the plasma. The deficiencies may be made good by increasing the salt intake by mouth, but where vomiting occurs resort to intravenous therapy may be essential. The potassium may also be deficient and this must be checked and rectified with care.

Bile salts which are lost must be replaced orally by tablets composed of a mixture of the various bile acids. Sometimes the bile itself is collected and fed by tube, though these methods seem messy and unpleasant and of doubtful value over the proprietary preparations now available.

There are absorptive difficulties when no bile has been reaching the intestines. The fat content of the stools is usually, though not necessarily, increased. Calcium ions are bound to fatty acids and form insoluble soaps depriving the body of an adequate calcium

intake. This is aggravated by the difficulty in absorbing the fat soluble vitamin D and one of the results is osteoporosis.

Vitamin K is also poorly absorbed resulting in a hypoprothrombinæmia and this must be made good to reduce the bleeding hazard. It is best given by daily injection though it does not follow that this will automatically restore the clotting facility to normal. A healthy liver is required to utilize the vitamin K and this may be so crippled as to be unable promptly to supply the deficient prothrombin. Restitution therapy should therefore be checked until normality is reached, so far as is possible: although surgery is generally reckoned to be safe with a prothrombin time (Quick) not more than twice that of the control. When an adequate coagulability cannot be achieved in this way resort to fresh transfusion may be required. At the same time coagulability and prothrombin time do not necessarily run parallel as prothrombin is not the only factor, according to modern work.

Liver damage is always an unknown quantity as the available liver tests give a poor estimate of the actual operative risk. Furthermore there is little that can be done to improve the liver condition while the obstruction is maintained. The liver cells are poor in glycogen and laden with fat. In such a state they are more vulnerable to any noxious influences and attempts at improvement are well worth while by means of alteration of diet to a high carbohydrate intake, where at least 80 per cent of calories come from this type of food. Intravenous glucose or preferably one of the invert sugar preparations may be required where a quicker effect is desired or the oral avenue is precluded. The deposition of glycogen may be encouraged by small insulin dosage. This therapy should be continued through the immediate post-operative period.

The infective factor must also be controlled so far as is possible in the presence of obstruction. The prevailing organism is B-coli and a suitable antibiotic should be selected. There is evidence that streptomycin does not find its way into the bile in effective quantity and chloromycetin or aureomycin may be more effective. Sulphonamides are probably better avoided as the possibility of post-operative anuria is always present in these liver cases.

At all times care must be taken with sedatives as impaired liver function enhances a patient's susceptibility to many of these drugs and a normal dose may prove fatal. Anoxæmia during anæsthesia is also dangerous to the liver cell.

The Approach

This will be determined to some extent by the previous scars on the abdomen and where possible an incision through normal anatomy is to be preferred. The paramedian, rectus split and Kocher incisions are well known. They should, however, be generous as exposure will be difficult and fine work at a depth requires a more easy access.

An alternative approach is sometimes used which has received much recent popularity for portocaval anastomosis where the liver is often large and very rigid. This takes advantage of the shelving under surface of the liver and aims to approach from a more posterior and lateral angle. The full incision was described by Satinsky of Philadelphia and is a thoraco-abdominal one passing usually through the eighth rib bed from the rib angle to the mid line (Fig. 77). This gives an excellent view of the hilum after the hepatic flexure of the colon has been mobilized and the diaphragm divided almost to the inferior vena cava and the liver displaced into the chest. It is not always necessary to

make the full incision and when occasionally it is made lower, perhaps through the tenth rib bed, the pleura need not necessarily be opened but can be pushed aside and mobilized prior to spreading the wound. The lower incision may be useful with very large livers.

Finding the ducts may be very difficult. The duodenum is often adherent to the under surface of the liver and must be mobilized before the region of the ducts can be exposed. The hepatic artery is a useful guide and can be felt pulsating parallel to the duct. The foramen of Winslow if at all definable is also a landmark. The upper end of the ducts

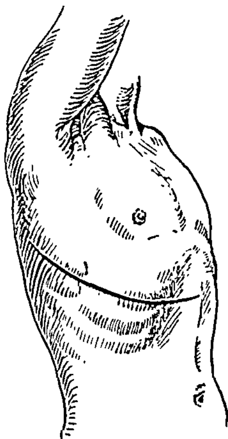


Fig 77 Satinsky incision Abdomino-thoracic approach to the porta hepatis through eighth rib bed from angle of rib to linea alba

is usually the easier to find as it is dilated and bulging. On occasions there may be no patent channel emerging from the liver in which case the intrahepatic area is explored with an aspirating needle in the hope of tapping a dilated upper end. The lower end may be entirely behind the duodenum or in the pancreatic substance where it is probable that it will have escaped previous injury at former operations. In these cases the duodenum and head of pancreas must be mobilized by an incision in the parietal peritoneum lateral to the second part of the duodenum and wiping the duodenum medially and forward and developing the plane posterior to the pancreatic head. Here the duct may be exposed, but often it is necessary to incise the pancreatic substance from behind in order to uncover a duct which is thus buried. It is permissible in difficult cases to incise longitudinally the anterior wall of the second part of the duodenum in order to

find the ampulla and pass a retrograde probe along the distal patent segment of the duct. The ampulla itself is not always easy to see (Fig. 78).

After a complete display of the duct remnants it will be possible to assess the degree and length of stricture and to plan some manœuvre to overcome it.

The various findings may be classified according to the following diagrams:

(1) A short stricture in common hepatic or common bile duct (Fig. 79 (*a*), (*b*)).

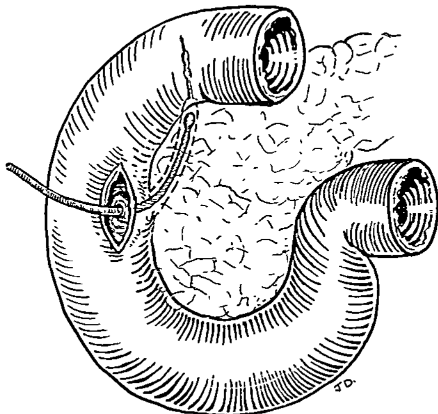


Fig 78 Retrograde bouginage of the strictured common duct when it cannot be found supraduodenally.

This is most usually found at the old site of entry of the cystic duct as this is the portion of duct most liable to trauma.

(2) A longer stricture is present at the upper end (Fig. 80 (*a*)).

(3) A long stricture is sited at the lower end (Fig 80 (*b*)).

(4) The ducts are obliterated except for a dilated upper end (Fig. 81 (*a*)).

(5) No duct exists between liver and duodenum (Fig. 81 (*b*)).

(6) The gall bladder is present above the stricture (Fig. 82).

Principles of Reconstruction. Certain principles have painfully emerged in the treatment of these conditions. They are:

(1) The sphincter of Oddi is the best mechanism yet designed to govern the entry of bile into the intestine and at the same time prevent the entry of intestinal contents into the biliary tract.

It should be preserved and made use of wherever possible. Hence it follows that end to end anastomosis of the duct is the ideal to be aimed at when continuity is once severed.

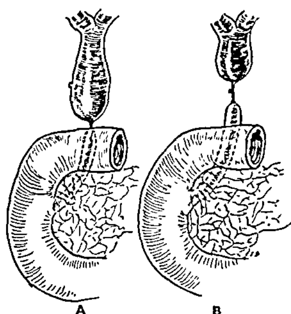


Fig. 79 Strictures of bile duct—localized—no gall bladder.

(a) Common bile duct.
(b) Common hepatic duct.

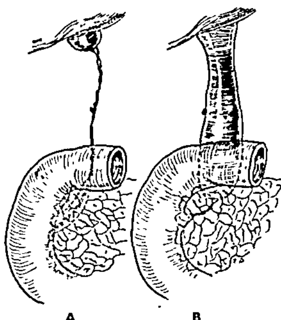


Fig. 80. Strictures of bile duct—no gall bladder.

(a) Long stricture—normal lower end.
(b) Long stricture—no normal duct distally (very unusual).

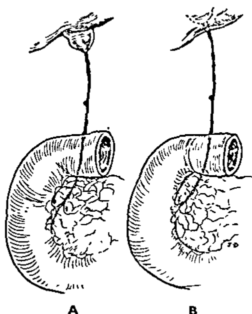


Fig. 81. Strictures of bile duct—no gall bladder.

(a) All duct obliterated except for a small extrahepatic segment.
(b) No normal duct visible.

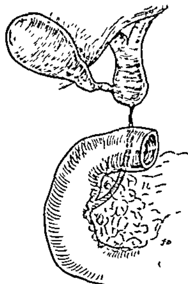


Fig. 82. Stricture below the cystic duct. Gall bladder present.

(2) Where the sphincter of Oddi can no longer be used a form of biliary-intestinal anastomosis should be employed which directs the food away from the biliary tract. Such a method is the Roux Y form of reconstruction (viz. Section III, p. 231—"Biliary Intestinal Anastomosis").

(3) The junction of duct to duct must be meticulously accurate to secure mucosal continuity at the earliest date in order to minimize the amount of granulation tissue which will eventually cicatrize.

(4) Internal splintage should always be employed and thus some provision made against mounting pressure within the duct. A T-tube answers these requirements and

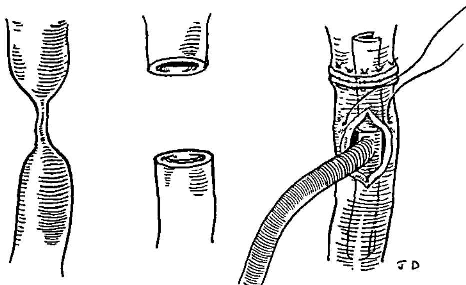


FIG 83 A short stricture is excised and continuity restored over a T-tube Interrupted sutures and separate T-tube exit.

should remain *in situ* until all danger of further stricture has passed. This may be six months or a year. The long arm of the tube should emerge at a point other than at the anastomosis.

(5) Any permanent form of internal tube is prone sooner or later to silt up with sludge and mud and become blocked. No material used so far is devoid of this danger. Any internal prosthesis should therefore ideally be temporary and capable of being withdrawn when its presence is no longer useful.

SHORT STRICTURES

These are the ones most easily excised and followed by end to end anastomosis of the ducts (Fig. 83). There must be no tension and interrupted sutures are preferable as there is no tendency to purse-string. Furthermore, as the stitches are usually of thread or silk in an infected field there may arise a small granuloma around each stitch and it is preferable that this should not be continuous around the entire duct wall as it is complete circumferential fibrosis which is most prone to severe contraction. The anastomosis is more easily made around a tube, which is usually the upper or lower limb of a T-tube. The horizontal arm is better brought out of the duct at a point away from the anastomosis in order not to delay healing or increase fibrosis at the critical point.

The T-tube ensures perfect drainage of the biliary tree during the critical early post-operative days when severe cholangiohepatitis is to be feared. It also acts as a splint preventing stenosis during the healing of the anastomosis and remains until this risk has disappeared. It is not known when such a time is reached. A minimum of three months is laid down though sometimes as long as a year is insisted upon. Surprisingly enough the retention of the tube for so long a period does not seem to damage the duct.

Many other prostheses have been tried in all shapes and materials, not only for duct to duct anastomoses but also those between duct and intestine. All are very prone to



FIG 84 Mobilization of duodenum and pancreatic head after the method of Cattell and Lahey In this way gaps following excision of strictures can be made good The intrapancreatic part of the duct is nearly always uninjured, but must be unburied

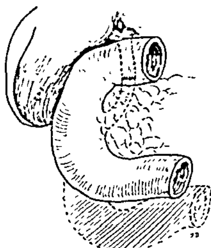


FIG. 85 The duodenum and pancreatic head raised to approximate the duct ends

blockage in periods ranging from a few months to several years. They should not on this account be designed to remain *in situ* but should pass on or be withdrawn after having served their purpose

The T-tube is retained by its emerging arm but can be withdrawn when no longer required. Those indwelling tubes with a flared upper end or collar are retained by stitches or held by the narrowness of the stoma for an unpredictable time. Others are fixed by a thread stitch emerging through the skin where it is tied to a surface button. The Pearse tubes have a phlange midway which projects from the duct and can be held by a stitch. Despite phlange and stitch these are often passed into the intestine. Soft and pliable tubes are kinder to the tissues and free from danger of pressure necrosis on neighbouring structures.

It is sometimes tempting to do a plastic repair of the Heineke-Mikulicz type when the stricture is localized. The objection to this method is that it does not excise the fibrosed area and should therefore be limited to those cases where there is no encircling fibrosis, but merely a narrowing due to part of the wall having been pinched off.

Longer Strictures but with Serviceable Ducts Above and Below

In these cases it is still possible to obtain a duct to duct anastomosis though at first sight there appears too wide a gap to be bridged. Cattell has emphasized the possibility of mobilizing the entire duodenum and pancreatic head (Fig. 83). In this way the head of the pancreas can be split upon its posterior surface so as to free the distal end of the duct which is frequently quite normal. Both the head of the pancreas and duodenum can

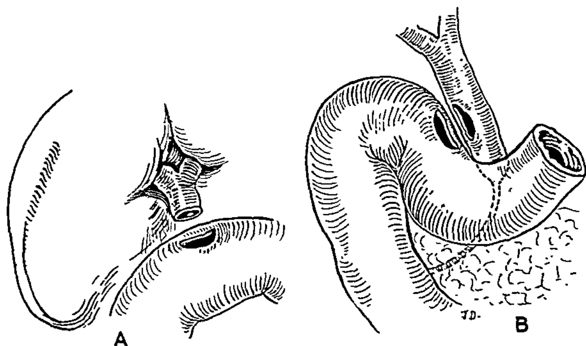


Fig. 86. Choledochoduodenostomy.
(*a*) End to side (*b*) Side to side.

then be raised even to the porta hepatis and the ducts placed in apposition without tension (Fig. 85).

Strictures which Extend into the Distal Common Duct

With this type of stricture there is no possibility of finding sufficient duct distally to attempt anastomosis. It is then necessary to abandon the sphincter of Oddi and make a fresh join of duct to intestine (viz. Section III—"Biliary Intestinal Anastomosis"). This can be done into duodenum or jejunum. The duodenum was always used until relatively recently when the jejunum became more popular. Anastomosis to duodenum can be end to side after division of the duct (Fig. 86 (*a*)) or side to side when the duct remains otherwise undisturbed (Fig. 86 (*b*)). This latter method is used when some small channel remains down through the normal sphincter.

The duodenum lies readily at hand and is of convenient thickness for stitching to the duct. It is relatively fixed, however, and cannot so easily as jejunum reach far into the porta hepatis when a really high anastomosis is necessary. Duodenal fistulæ do occasionally follow and are much more unpleasant than a similar one from a defunctioned jejunum. Apart from these considerations there is the much discussed incidence of recurrent cholangitis which may be due to regurgitation of food into the biliary tract. This can be largely obviated where an entero-anastomosis (Fig. 87 (*a*)) or a jejunal Y is used (Fig.

The T-tube ensures perfect drainage of the biliary tree during the critical early post-operative days when severe cholangiohepatitis is to be feared. It also acts as a splint preventing stenosis during the healing of the anastomosis and remains until this risk has disappeared. It is not known when such a time is reached. A minimum of three months is laid down though sometimes as long as a year is insisted upon. Surprisingly enough the retention of the tube for so long a period does not seem to damage the duct.

Many other prostheses have been tried in all shapes and materials, not only for duct to duct anastomoses but also those between duct and intestine. All are very prone to



FIG 84 Mobilization of duodenum and pancreatic head after the method of Cattell and Lahey In this way gaps following excision of strictures can be made good The intrapancreatic part of the duct is nearly always uninjured, *but must be unburied*

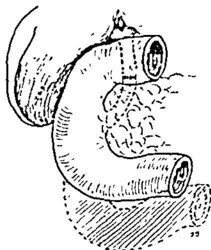


FIG. 85 The duodenum and pancreatic head raised to approximate the duct ends

blockage in periods ranging from a few months to several years. They should not on this account be designed to remain *in situ* but should pass on or be withdrawn after having served their purpose.

The T-tube is retained by its emerging arm but can be withdrawn when no longer required. Those indwelling tubes with a flared upper end or collar are retained by stitches or held by the narrowness of the stoma for an unpredictable time. Others are fixed by a thread stitch emerging through the skin where it is tied to a surface button. The Pearse tubes have a phlange midway which projects from the duct and can be held by a stitch Despite phlange and stitch these are often passed into the intestine. Soft and pliable tubes are kinder to the tissues and free from danger of pressure necrosis on neighbouring structures.

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an inch leaving the mucosa and submucous vessels only. This layer is fitted about a conical prosthesis and pushed into the liver defect as a mucosal graft (Fig. 90).

A third alternative is that described by Longmire in which the left hepatic duct is found by amputating about two-thirds of the left lobe of the liver and anastomosing this after a little dissection to a jejunal limb. This drains the right lobe in a retrograde direction up the remaining left hepatic duct and so into the intestine. The accompanying

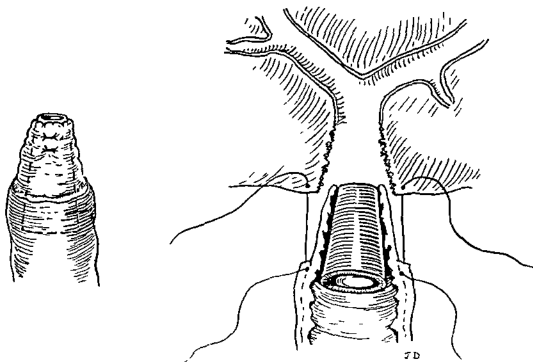


FIG 90 Implantation of jejunal mucosal cone into cored out liver aperture when no extrahepatic ducts are found

branch of the right hepatic artery is secured close to the duct and the hæmorrhage from the cut surface is controlled by long mattress sutures (Fig. 91).

Finally it occasionally happens that a gall bladder is present and can be utilized for bypassing a common duct stricture. It must, of course, be shown to communicate with the dilated part of the ducts. The gall bladder can be anastomosed to stomach, duodenum or jejunum. The stomach is not favoured because of its high mobility, the muscularity of its walls which do not match the wall of the gall bladder, and the greater power of its contractions, which may encourage reflux.

Such a happy solution to the problem can only be expected in rare cases of spontaneously occurring stricture or in those due to chronic pancreatitis.

Bridging of defects by tubes is becoming increasingly unpopular in view of the extremely poor results obtained. It was hoped that the prosthesis might act as a scaffold around which new biliary epithelium would grow. Such has seldom proved to happen. The tubes are difficult to maintain in position and sooner or later become choked with biliary detritus.

Results. These depend very much on the individual findings. The outlook materially worsens with the number of preceding operations; with the length of the strictures and

No Ducts Visible Below the Liver

The results are the poorest where no ducts are visible beneath the liver. These cases should not be abandoned, however. The old method was to incise into the liver hilum thus creating an external fistula if this did not already exist. Sometimes a drain was left in the peritoneum until it ulcerated through the bowel and made an internal fistula. This is a very uncertain method. Alternatively the fistulous track was later excised and

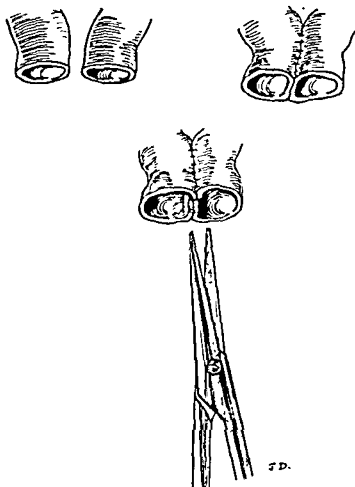


Fig 89 Cattell's method of uniting right and left hepatic ducts to form one channel for anastomosis.

implanted into the intestine, as first practised by Williams. But as even the external fistula is an unsatisfactory drainage, always trying to close, there is no reason why the same track should be any better internally. After dissection the track is apt to be rather avascular and seldom provides permanent adequate decompression. Nevertheless there are many successful cases recorded. It is probably most likely to succeed when the fistulous track is discarded with the exception of the last half inch and over this a limb of jejunum is sewn to the liver under-surface.

Cole described a modification of Hoag's technique whereby he excises a cone of liver tissue leading to the dilated intrahepatic channels. Into this he addresses a similar cone of jejunum, from which the terminal muscular coat has been removed for about

contiguous viscera have enjoyed temporary notoriety only to be abandoned in most cases in the experimental phase. The greatest improvement no doubt lies in the prevention of injuries to the duct at operation, the early removal of all common duct stones and the energetic treatment of recurring cholangitis.

References

(1) Allen, A. W. (1945) *Ann. Surg.* **121**, 412.
(2) Cattell, R. B. (1943) *Surg. Clin. N. Amer.* **23**, 701.
(3) Cattell, R. B. (1947) *J. Amer. med. Ass.* **134**, 235.
(4) Cole, W. H. (1949) Operative Technique, Vol. 1. *Appleton Century Crofts.*
(5) Flickinger, F. M. and Masson, J. C. (1946) *Surg. Gynec. Obstet.* **83**, 24.
(6) Glenn, F. and Hays, D. M. (1952) *Surg. Gynec. Obstet.* **94**, 283
(7) Gray, H. K. [illegible]
(8) [illegible] Oxford.
(9) [illegible]
(10) [illegible]
(11) [illegible]
(12) Laney, F. H. and Pyrtek, L. J. (1950) *Surg. Gynec. Obstet.* **91**, 25.
(13) Longmire, W. P. and Sanford, M. C. (1948) *Surgery.* **24**, 264.
(14) McArthur, L. L. (1923) *Ann. Surg.* **78**, 129.
(15) O'Malley, R. D., Aufses, A. H. and Whipple, A. O. (1951) *Ann. Surg.* **134**, 797
(16) Pearce, A. E. et al. (1951) *Ann. Surg.* **134**, 808
(17) Pearse, H. E. (1942) *Ann. Surg.* **115**, 1031.
(18) Peterson, L. W. and Cole, W. H. (1948) *Arch. Surg.* **56**, 445.
(19) Satinsky, V. [illegible]
(20) Sedgewick [illegible]
(21) Shea, P. C. [illegible]
(22) Soresi, A. [illegible] *Amer. J. Surg.* **82**, 671.
(23) Sullivan, A. G. (1909) *J. Amer. med. Ass.* **53**, 774.
(24) Turner, G. Grey (1944) *Lancet*, **1**, 621.
(25) Walters, W. [illegible]
(26) W [illegible] 150.
(27) W [illegible]

CONGENITAL ANOMALIES AND MALFORMATIONS

Anatomy

THE normal anatomy of the gall bladder and biliary ducts may be culled from any anatomy book. It is the departures from normal anatomy which most concern the surgeon in this field owing to the danger of injury to vital structures when their arrangement deviates from what is anticipated. The surgeon must be aware of the congenital abnormalities so frequently encountered in this area. So common is some departure from the average that it is scarcely possible to describe a normal arrangement of arteries and ducts, though the ducts are more constant than the arteries.

Anatomical Variations

BILIARY CHANNELS

(*a*) GALL BLADDER. This may be absent or hidden within the liver. It may be double or bicornuate. It may be entirely invested with peritoneum or provided with a mesentery or applied to the under surface of the liver by a broad extraperitoneal attachment. There

their proximity to the liver. It is adversely affected by the duration and depth of jaundice which is usually a reflection of the state of the liver cells. The experience of the surgeon counts for much.

Despite this, reports claim as high a rehabilitation rate as 60–80 per cent, with a mortality of under 5 per cent. The condition of the unsuccessful 20 per cent is usually

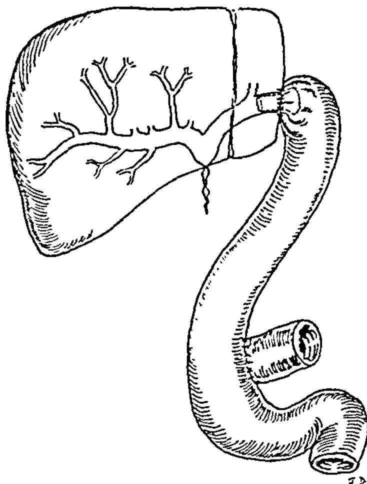


FIG 91. Method of Longmire for securing biliary-intestinal drainage by amputation of left lobe of liver and utilization of left intrahepatic duct.

aggravated by the operation and most of those leaving hospital are dead within two years.

Of the fatalities liver failure stands highest as a cause of death. Hæmorrhage assumes a much less significant role, though biliary peritonitis still claims its victims despite almost universal drainage. The liver failure usually takes the form of a severe hepatitis with cholæmia or frank cellular necrosis.

The Future. It is difficult to see what lies ahead in the treatment of these conditions. The search for a viable biological tube which will function in the place of the missing duct has continued since the optimistic reports of Horsley in 1918 using free venous grafts. Since then all things animal, vegetable, and mineral have been tried with disappointing results. Arteries, veins, ureters, appendices, peritoneal and mucosal flaps from

Arteries

(*a*) Hepatic Artery. This is normally a branch of the cœliac axis giving off the gastroduodenal artery before turning upwards in the lesser omentum towards the liver. It runs parallel and medial to the common bile duct and divides into left and right hepatic branches at about the junction of cystic and hepatic ducts—both branches lying on a plane behind the corresponding hepatic ducts.

The following arteries have been found on occasions to pass anterior to the bile ducts—the gastroduodenal, the common hepatic, the right hepatic, the cystic, and the right gastric, and the superior pancreatico-duodenal. The right hepatic artery arises rarely from the superior mesenteric and reaches the liver separately on a plane posterior to the bile duct. An accessory right hepatic artery is sometimes present. The right hepatic artery when arising normally passes behind the common hepatic duct in approximately 70 per cent of cases and anterior to it in the remainder. In either position it forms a very close relationship to the gall bladder neck, especially when tortuous.

(*b*) The Cystic Artery. This arises from the right hepatic artery in 95 per cent of cases at a point to the right of and posterior to the common hepatic duct. In the remainder it may arise from the common hepatic, the left hepatic or the gastroduodenal. It passes anterior to the ducts in approximately 15 per cent of cases. There may be more than one cystic artery.

CONGENITAL OBSTRUCTION OF BILE DUCTS

This condition is presumably due to a failure of development during fœtal life in which the one time solid state of the ducts persists. The occurrence is rare but a study of postmortem material indicates that surgical relief, which depends on the site of the atretic area, should be feasible in some 20 per cent of cases. There may be no more than a stenosis but in other cases there is obliteration of the lumen over a whole or part of the extrahepatic biliary system. The ducts and gall bladder may be represented by fibrous strings, but in those cases where the lower ducts alone are atretic distended channels are found between liver and the affected area.

The liver shows advanced biliary cirrhosis and there may be evidence of portal hypertension.

Clinical Findings. Such children are born with an obstructive jaundice of a progressive, complete, and unrelenting type though the icterus may be misunderstood during the first few weeks of life. The stools are consistently acholic and the urine dark with its content of bile pigments The jaundice may reach a very severe intensity and most patients will die during the first few months if the jaundice is unrelieved. Death is due to intercurrent infection, marasmus or hæmorrhage.

The liver is considerably enlarged and not quite smooth and this with a slight splenomegaly and ascites give a distended abdomen. These children are usually retarded and intolerant of fats. It is a matter of some interest that in this type of obstructive jaundice alone the alkaline phosphatase is within normal limits.

Diagnosis. Other causes of jaundice must be considered. These are:

(1) Icterus neonatorum.

is often a mesenteric fold passing to the mesocolon or duodenum. The position of the gall bladder is not constant.

(*b*) CYSTIC DUCT. This varies considerably in length and width. It may open at any point upon the hepatic or common bile ducts at times running parallel for some distance before terminating into the common duct or even duodenum. It may enter anteriorly, posteriorly or even medially in about 10 per cent of cases. When entering upon the left side it usually passes behind the common duct and may completely encircle it. These modes of junction are classified as angular (63 per cent), parallel (20 per cent), or spiral (17 per cent).



FIG 92 Cholangiogram through a T-tube, showing the common duct and its termination outlined and reflux of dye up the pancreatic duct

(*c*) THE RIGHT AND LEFT HEPATIC DUCTS. These join extrahepatically in nearly all cases in which the liver is not enlarged. There is sometimes a third hepatic duct from the quadrate lobe. The hepatic ducts may be multiple. The order of structures from anterior to posterior in the hilum is duct-artery-vein. There is rarely (4 per cent) an artery passing anterior to the hepatic ducts or their junction. *The hilar margin of the quadrate lobe* is the landmark for the junction of the ducts in nearly all cases in which the anatomy is otherwise obscured. The cystic duct may enter the right, or more rarely the left hepatic duct.

(*d*) COMMON HEPATIC DUCT. The length of this is determined by the point of entry of the cystic duct and is thus sometimes nonexistent.

(*e*) COMMON BILE DUCT This again varies inversely with the length of the common hepatic duct. Where the cystic duct runs parallel to the common bile duct the two are often bound together in a common sheath so that the arrangement may be invisible from without. The cystic duct rarely extends to within half a centimetre of the ampulla of Vater. In 20 per cent of cases there is thus no supra-duodenal common bile duct.

Choledochocele and congenital obliteration of the bile ducts are described elsewhere.

The manner of termination of the common bile duct is possibly of importance in the ætiology of pancreatitis. The pancreatic duct and common bile duct unite to form a single channel before entering the duodenum in a proportion of cases—figures vary from a 3·5 to 77 per cent incidence. The important point is perhaps the incidence of a common sphincter or a final common channel rather than a common termination and hence the varying figures (Fig 92)

Accessory bile ducts are present in about 15 per cent of cases. They mostly come from the right lobe and enter the right hepatic, common hepatic or cystic ducts. They may also enter the gall bladder. Sometimes they pass from duct to the same duct at a lower point.

or only slightly enlarged. The dilated part may reach enormous sizes but is commonly as large as an apple. It occurs in females in 80 per cent of cases and the majority begin to experience symptoms in the first decade and to come to treatment in the second though some patients present as late as the fourth decade. The condition has been found in the fœtus.

The cyst has a wall of connective tissue with an incomplete epithelial lining showing signs of inflammation. The inner surface may be rough as a result of infection and precipitated biliary detritus and the outer wall adherent to peritoneum and other structures. It usually involves only the common bile duct but may extend upwards or downwards so that the left and right hepatic, cystic or pancreatic ducts enter directly into the cyst. The contents vary from normal or infected bile to clear fluid.

A variety of findings in the duct below the cyst have been described, most of which are probably secondary to the size and weight and infection of the enlarging cyst. Thus valves, kinks, and stenoses are recorded, but all are agreed that often there is no hint of any form of obstruction and a probe may easily be passed. This is reminiscent of the congenital hydronephrosis and Hirschsprung's disease and invites the theory that there is some underlying congenital neuromuscular inco-ordination, perhaps associated with a local weakness of the duct wall. This idea is perhaps strengthened by the frequency of other congenital abnormalities in the same individual.

Complications. These may be listed:
(1) Infection within the cyst
(2) Ascending cholangitis.
(3) Stone formation
(4) Cirrhosis of the liver.
(5) Rupture.
(6) Thrombosis of portal vein.

Clinical Features. Pain is the most frequent symptom generally arising in childhood and going back over several years. It may be slight and felt in the epigastrium or right hypochondrium with occasionally severe bouts of colic suggestive of biliary disease. The pain may be related to meals and there is frequently an associated digestive disturbance with vomiting on occasions. Mild jaundice of an obstructive type is present in 65–90 per cent of cases. It is usually intermittent and may be associated with pyrexia or even rigors.

A tumour is palpable in the upper abdomen and towards the right in 60 per cent of cases. It may be tender and is smooth and globular. It may seem to be part of the liver, though the liver edge should be palpable above the mass. It is resonant if the colon lies over it. It moves with respiration to an extent less than does the liver and can be manipulated from side to side more freely than up and down. It does not lift from the loin. The liver itself, and rarely the spleen, may be slightly enlarged. The consistency varies, and sometimes in relation to meals. At times it may feel like a firm solid and at times is recognizably cystic. This feature is of considerable diagnostic assistance. To these signs may be added in infected cases the peritonism and toxicity and leucocytosis as seen in cholecystitis.

Diagnosis. The combination of pain in childhood, jaundice and a mass in the hypochondrium are not often encountered in any other condition and when the mass

This is a transient, mild and gradually improving jaundice, unassociated with hepatomegaly, steatorrhœa or choluria.

(2) Icterus gravis.

These infants do not often survive long enough to be confused with biliary atresia. There is an erythroblastosis and may have been obstetric abnormalities to indicate such a diagnosis.

(3) Hæmolysis of septicæmia.

The infective element is usually apparent and the urine contains no pigment while the stools are coloured.

(4) Congenital syphilis.

(5) Obstruction of the bile ducts by plugs.

These cases are indistinguishable from atresia unless there has been some evidence of intermittence of the obstruction.

(6) Atresia of duodenum, above and below the ampulla.

Treatment. The inevitable outcome is certain death unless relief can be obtained surgically. Only about 20 per cent of cases can be corrected and of these the operative mortality will claim one in three. Even on these terms laparotomy cannot be denied wherever the diagnosis is definite. The optimum age for operation is between one and three months.

Preoperatively the child's condition can be improved by small transfusions and vitamin K, and a low fat diet may help to improve the state of nutrition.

General anæsthesia and an adequate right paramedian incision are necessary. It is not easy to interpret the state of the ducts as they are so small that the presence of a lumen may sometimes only be proved by the injection of saline into the gall bladder when this itself is present and has a lumen. This manœuvre will serve to identify and relieve those cases where the obstruction is due to plugs of mucus and no atresia exists. It will also distend those patent parts of the duct system in contrast to the imperforate areas.

Where no distended patent channel exists below the liver the case is considered inoperable.

If the gall bladder contains bile it is evidence of an intact cystic and common hepatic duct. The obstruction may be higher than the origin of the cystic duct and still be amenable to surgery where the distended common hepatic duct is visible.

The operation of choice is choledocho-duodenostomy or choledocho-enterostomy though it is tempting to employ the gall bladder for the anastomosis when this communicates with the liver With higher obstruction hepatico-duodenostomy may be required. The anastomosis should be made over a soft polythene tube with a single row of interrupted silk sutures. The tubing eventually passes on providing it is no longer than necessary to support the anastomosis The area is drained and the wound sewn up with especial care as distension and malnutrition may lead to a burst abdomen.

CYSTIC DILATATION OF THE BILE DUCTS

This is an entity quite distinct from the acquired generalized dilatation of the entire biliary tree secondary to obstruction. It differs in that one part, usually the supraduodenal portion, dilates out of all proportion to the rest of the ducts which are normal

Congenital obstruction:
(1) Beavan, T. E. D. and Duncan, G. W. (1946) *Brit. J. Surg.* 33, 378.
(2) Ladd, W. E. (1935) *Ann. Surg.* 102, 742.
(3) Thomson, J. (1891) *Edinb. med. J.* 37, 523.

Cystic dilatation:
(1) Brunschwig, A. and Clark, D. E. (1941) *Surgery,* 10, 553.
(2) Gross, R. E. (1933) *J. Pediat.* 3, 730.
(3) Irwin, S. T. and Morison, J. E. (1944) *Brit. J. Surg.* 32, 319.
(4) [illegible]
(5) [illegible]
(6) [illegible] 1, 912.
(7) [illegible]
(8) [illegible] 77.

TUMOURS OF THE GALL BLADDER AND BILE DUCTS

Gall Bladder

Benign. Benign tumours of the gall bladder, such as papilloma, are extremely rare and are usually reported as a postmortem finding. They are distinct from the polypoid conditions of the mucosa in cholesterosis and much inconclusive controversy has surrounded their possible role as precancerous lesions.

Malignant. Carcinoma of the gall bladder occurs in later life in the proportion of four females to one male and is encountered in from 1–2 per cent of all operations on the gall bladder. It is occasionally an unexpected finding either at cholecystectomy, when its presence may escape the surgeon, or at postmortem following death from carcinomatosis.

Pathology. There are described four types of carcinoma, according to their microscopical appearances.

(1) Scirrhus—diffusely infiltrating.
(2) Papillary—growing into the lumen of gall bladder and ducts.
(3) Colloid—with early peritoneal spread.
(4) Squamous—arising in metaplastic epithelium.

This differentiation carries no clinical significance. The carcinoma remains confined to the gall bladder for a very short time and quickly spreads to other organs, especially by direct extension into the liver, duodenum and stomach, colon and peritoneum. Lymphatic spread soon involves the cholecystic gland and others within the porta hepatis. Blood stream spread occurs late

Once beyond the gall bladder wall the lesion very soon becomes inoperable owing to the number of irremovable structures implicated. The ducts may become obstructed either from within by a papillomatous lesion or from without by pressure of a gland or by infiltration of the cancerous cells. In this way mucocœle, empyema, acute cholecystitis and obstructive jaundice may complicate the picture.

Carcinoma seldom arises in an otherwise normal gall bladder. It is computed that here is an antecedent chronic cholecystitis or gall stone in from 80–90 per cent of cases.

has such characteristic features the diagnosis is not difficult if the condition is borne in mind. Its rarity, however, is the main reason why a preoperative diagnosis is not more frequently made. At operation the findings are difficult to interpret if the surgeon is not familiar with the condition.

Radiologically the rounded outline of the soft tissue swelling may be visible. There may be calcification in the cyst wall; a barium meal may show displacement of duodenum and colon downwards and to the left, and a cholecystogram reveal a concentrating gall bladder moulded around the top of the cyst.

The tumour may be mistaken for:

(1) An acute cholecystitis with mass.
(2) A mucocele or empyema of the gall bladder.
(3) A hydronephrosis.
(4) A malignant tumour of kidney, suprarenal or liver.
(5) A polycystic kidney.
(6) A solitary renal cyst.
(7) A hydatid cyst of the liver.
(8) A pancreatic cyst.

The symptoms without a tumour will almost certainly lead to a diagnosis of cholelithiasis, especially in an adult.

Treatment. The condition is dangerous and produces much chronic misery and in general requires operative treatment. A large proportion are found at operation and these should all be dealt with. There may be some hesitation to insist on surgery in those in whom the diagnosis is reached without laparotomy in view of the high published mortality of 60 per cent. These are old figures, however, including many outmoded forms of operation now known to be dangerous. Present day mortality for correct operative treatment should not exceed 10 per cent, especially if it is carried out before complications arise. The safest and most effective treatment is to anastomose the cyst to the duodenum to which it is nearly always closely applied and to resist temptation to excise the cyst or drain it externally as these two manœuvres are associated with a high mortality. After the duodenocystostomy the cyst shrinks considerably in the course of time and although an occasional patient continues with attacks of cholangitis this is exceptional and the majority of the survivors are symptom free. For this reason it does not seem necessary, especially in children, to go to the lengths of fashioning a Roux anastomosis of jejunum with which to drain the cyst If there is an acute inflammation of the cyst it would be wise to insert a T-tube into the normal duct above the cyst or failing this carry out a cholecystostomy in addition to the duodenocystostomy.

References

Anatomy·
(1) [illegible]
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usually impossible to determine the precise point of origin once the lesion has advanced. Such a determination has no profound importance to the surgeon, to whom the problem of treatment is identical when the growth originates within this area.

The possible sites include: the bile duct, the ampulla of Vater, the duodenal papilla, the pancreatic duct, the head of the pancreas or the duodenum itself close to the papilla. The pancreatic head and its lymphatics are implicated almost from the start, so that the problem of surgical removal varies but little in each case. The cytology does not provide a sure method of sorting the different types, being mostly mucous secreting adenocarcinomata.

Lesions in all sites quickly produce a back pressure within the ducts leading to obstructive jaundice with all its accompaniments, and this often at a time when the ducts will still admit a probe. The obstructed ducts dilate even up to the intrahepatic biliary radicals (hydrohepatosis) giving rise to hepatomegaly, centrilobular cellular atrophy and later biliary cirrhosis in a few cases. The state of the gall bladder depends on the site of the obstruction and is usually enlarged with distal lesions, unless previously fibrosed. Should the cystic duct become blocked by growth a mucocœle or cholecystitis with empyema may result. Ulceration into the duodenum is common with the lower lesions, resulting in melæna and anæmia or even duodenal stenosis. Ascites is a late finding.

Clinical Features. The patients usually present themselves with obstructive jaundice and although this according to classical teaching is unvarying and painless the reverse is often true. Biliary colic is rare, but pain is often felt over the liver area and in the back which is more fixed and less severe than colic The jaundice, which may be associated with chills varies in degree but very seldom completely disappears. Appetite is poor and flesh is lost. Bleeding into the duct or more usually into the duodenum drains the iron from the body and occult blood or melæna are often present in the stools. The abdomen often shows nothing but the bile staining, a large liver and perhaps an hydropic gall bladder. In short the patient at the start is relatively well, with an obstructive jaundice without evidence of cholecystitis The condition is virtually indistinguishable from carcinoma of the pancreas, though some help may come from the newer techniques of cholangiography when the lesion lies more proximally than the pancreas

Treatment. Laparotomy is essential in all cases.

The treatment carried out will depend on the findings at laparotomy, especially the site of the tumour and the extent of its spread. The aims are to remove all the cancerous cells and restore biliary continuity, or failing this to provide internal biliary drainage.

Palliative Procedures. These are indicated when resection is impossible:

(a) *Cholecystenterostomy* can be carried out when a gall bladder is present and the lesion is distal to the junction of the cystic with the common hepatic duct.

(b) When the lesion is above this point and not resectable owing to its high position with no normal duct above, or owing to the extent of metastasis, the duct below the obstruction is opened. An attempt is made to canalize the diseased area and insert a permanently indwelling polythene tube either in the form of a long T, which can be kept free by irrigation, or a simple straight tube over which the duct is closed. Full palliation for periods longer than a year have thus been achieved, though there is always the possibility of silting in the latter type of prosthesis

Clinical Features. There is no constant syndrome associated with this condition, which is usually unsuspected. There is in the majority of cases a previous history suggesting gall bladder disease, with dyspepsia, colic or jaundice. The familiar pattern of such suffering changes not long before the patient seeks advice. This change may be in the character of the pain which becomes more unrelenting, fixed and unaffected by meals. Jaundice may have occurred or a mass been felt by the patient. In other cases the symptoms are those of an unlocalized intra-abdominal malignancy, with wasting, ascites and hepatic or peritoneal deposits.

The tumour is often palpable as a hard irregular mass inseparable from the inferior margin of the right hepatic lobe. Moving on respiration, it is dull to percussion and only slightly tender. There may be an audible rub and the distended dome of a mucocœle is occasionally present. Free fluid or omental masses may be detected and the liver surface present the umbilicated bosses of multiple intrahepatic metastases. The urine will contain bile when the patient is icteric.

Treatment. The fact that so many carcinomas arise in chronically inflamed gall bladders is often given as a reason for cholecystectomy before such a complication, which is thus theoretically preventable, arises. Although this is true enough, carcinomatous degeneration is nevertheless rare considering the frequency of gall stones and gall bladder disease.

In the removal of a carcinomatous gall bladder the scope of the operation cannot be *extended beyond the procedure of routine cholecystectomy, with the exception* of the removal of an adjacent liver wedge or the picking off of a few glands in the lesser omentum. For this reason the results of excision are almost uniformly poor and a cure is very seldom obtained. On the contrary the rapidity of spread and rate of growth is very often accelerated by such an undertaking. Almost all the satisfactory results are to be found amongst those cases in which a carcinoma is removed unwittingly in a gall bladder believed only to harbour stones and inflammation.

Nevertheless where the prospect of resection seems reasonable it should be attempted for the sake of the few.

Carcinoma of the Bile Ducts

Malignant tumours of the biliary passages are rare though they occur at least half as frequently as those of the gall bladder itself and according to some authorities more frequently than carcinoma of the pancreas.

Pathology. (*a*) Intrahepatic The cholangiomata have already been mentioned

(*b*) Extrahepatic. This seems to be a different disease to the gall bladder carcinoma in so far as the close association with gall stones no longer applies, and the sex incidence is reversed. Carcinoma of the ducts occurs in the ratio of 3 men to 2 women.

The lesions occur with increasing frequency as the duct is descended and form plaque-like areas extending in the substance of the duct wall. They have been divided into nodular and papilliferous forms though the structure is usually that of a columnar adenocarcinoma. The growth spreads up and down the duct system and to the other structures in contact with it Local lymphatic glands and the liver substance are invaded by metastasis.

(*c*) Intrapancreatic This includes all tumours arising in that part of the duct distal to the first part of the duodenum and includes man

usually impossible to determine the precise point of origin once the lesion has advanced. Such a determination has no profound importance to the surgeon, to whom the problem of treatment is identical when the growth originates within this area.

The possible sites include: the bile duct, the ampulla of Vater, the duodenal papilla, the pancreatic duct, the head of the pancreas or the duodenum itself close to the papilla. The pancreatic head and its lymphatics are implicated almost from the start, so that the problem of surgical removal varies but little in each case. The cytology does not provide a sure method of sorting the different types, being mostly mucous secreting adenocarcinomata.

Lesions in all sites quickly produce a back pressure within the ducts leading to obstructive jaundice with all its accompaniments, and this often at a time when the ducts will still admit a probe. The obstructed ducts dilate even up to the intrahepatic biliary radicals (hydrohepatosis) giving rise to hepatomegaly, centrilobular cellular atrophy and later biliary cirrhosis in a few cases. The state of the gall bladder depends on the site of the obstruction and is usually enlarged with distal lesions, unless previously fibrosed. Should the cystic duct become blocked by growth a mucocœle or cholecystitis with empyema may result. Ulceration into the duodenum is common with the lower lesions, resulting in melæna and anæmia or even duodenal stenosis. Ascites is a late finding.

Clinical Features. The patients usually present themselves with obstructive jaundice and although this according to classical teaching is unvarying and painless the reverse is often true. Biliary colic is rare, but pain is often felt over the liver area and in the back which is more fixed and less severe than colic. The jaundice, which may be associated with chills varies in degree but very seldom completely disappears. Appetite is poor and flesh is lost Bleeding into the duct or more usually into the duodenum drains the iron from the body and occult blood or melæna are often present in the stools The abdomen often shows nothing but the bile staining, a large liver and perhaps an hydropic gall bladder In short the patient at the start is relatively well, with an obstructive jaundice without evidence of cholecystitis The condition is virtually indistinguishable from carcinoma of the pancreas, though some help may come from the newer techniques of cholangiography when the lesion lies more proximally than the pancreas.

Treatment. Laparotomy is essential in all cases

The treatment carried out will depend on the findings at laparotomy, especially the site of the tumour and the extent of its spread. The aims are to remove all the cancerous cells and restore biliary continuity, or failing this to provide internal biliary drainage.

PALLIATIVE PROCEDURES. These are indicated when resection is impossible:

(*a*) *Cholecystenterostomy* can be carried out when a gall bladder is present and the lesion is distal to the junction of the cystic with the common hepatic duct.

(*b*) When the lesion is above this point and not resectable owing to its high position with no normal duct above, or owing to the extent of metastasis, the duct below the obstruction is opened. An attempt is made to canalize the diseased area and insert a permanently indwelling polythene tube either in the form of a long T, which can be kept free by irrigation, or a simple straight tube over which the duct is closed. Full palliation for periods longer than a year have thus been achieved, though there is always the possibility of silting in the latter type of prosthesis.

(*c*) The creation of an external biliary fistula by incising into the porta hepatis and allowing bile to drain externally with perhaps a later insertion of the track into the bowel. This is an uncertain and dangerous method and liable to increase the patient's misery.

RESECTION. (*a*) *Suprapancreatic Lesions.* When the upper limit of the growth is within reach and there are no distant metastases resection is possible in the early stages.

The operation can be divided into two stages.

(i) *The resection* which should include the growth and at least 1 cm. of normal duct above and below the lesion. All lymphatic glands along with the fat and supportive tissues of the lesser omentum and porta hepatis are removed. The gall bladder is included in the resection when the cystic duct is involved.

(ii) *The restoration of continuity.* The problems are akin to those encountered in the reconstruction of the biliary tract after excision of strictures (q.v.). End to end anastomosis is the ideal and may be achieved over a T-tube and by mobilizing and raising the pancreatico-duodenal unit in order to secure approximation. Failing this, with a low resection continuity may be restored by a cholecystenterostomy or choledochenterostomy, but these are less satisfactory.

Such operations fall short of the ideal principles of carcinoma surgery, being limited, piecemeal, and patchy, but to some extent are justified by occasional success and the earliness of the lesion.

(*b*) *Intrapancreatic lesions.* There is little profit in separating from the point of view of surgical excision carcinomata arising in the terminal duct, ampulla, papilla, neighbouring duodenum or in the pancreatic duct. The pancreas and duodenum are often invaded in all lesions and the lymphatic drainage of the area is so intimately bound up with the pancreatic head and duodenum that a complete clearance must include these structures. The exact site of origin is seldom known and a more or less standard pancreatico-duodenectomy has now been worked out which is applicable to lesions within this area. It is true that lesser operations are done and not without success, such as a transduodenal resection of the papilla, but this is not really adequate as an attempt to eradicate a carcinomatous lesion and should be used less and less with increasing proficiency with the more radical technique. The mortality of the larger operations is in some series less than that of the more limited.

It may well be that such lesions as we are now considering will remain the only indication for radical pancreatico-duodenectomy as the results of this operation for carcinoma of the pancreas proper are increasingly disappointing.

For a description of the radical operation see carcinoma of pancreas.

TRANSDUODENAL EXCISION OF THE PAPILLA. This has been carried out for very early lesions of the papilla The second part of the duodenum is incised longitudinally in the anterior wall and the papilla with its growth identified and raised through the duodenal incision by three guy sutures inserted into the posterior duodenal wall just outside the area of intended resection. The duodenal wall around and including the tumour and as large a margin as possible is then incised so that this segment remains attached by the pancreatic and bile ducts only. These are drawn through the hole as far as possible and caught with a suture before being severed. Their cut ends are then anastomosed by interrupted silk sutures to the duodenal mucosa and muscle at the rim of the hole. The operation usually fails because the extent of the resection is severely limited.

Results. The operative mortality is in the region of 30 per cent for both operations.

Palliative Procedures. Internal biliary drainage is associated with a higher mortality than might have been expected and figures as high as 65 per cent have been quoted. This in general reflects upon the advanced state of disease of such patients upon whom this is the procedure of choice.

The results in survivors are good with a relief of jaundice and irritation, although the average expectation of life is under two years. Several cases passing the five year mark are within the experience of most surgeons.

Transduodenal Excision. The mortality is high, chiefly from leakage of bile and and pancreatic juice and 35–50 per cent operative death rate may be expected. The five year survival rate is almost nil.

Radical Pancreatico-duodenectomy. There is some evidence that the operative mortality is less when this operation is done for a papillary cancer rather than one of the pancreas proper. The operative death rate probably lies between 10 and 25 per cent and as far as can be discovered the five year survival rate may lie between 25 and 40 per cent.

References

Gall bladder:

(1) Cooke, L., Jones, F. Avery and Keech, M. K. (1953) *Lancet* 2, 585
(2) [illegible]
(3) [illegible]
(4) [illegible]
(5) [illegible]
(6) [illegible]

Bile ducts:

(1) Brunschwig, A (1948) *J. Amer. med. Ass.* 136, 28.
(2) Cattell, R. B. (1945) *New Engl. J. Med.* 232, 521
(3) [illegible]
(4) [illegible]
(5) Hunt, V. C. and Budd, J. W (1935) *Surg. Gynec. Obstet.* 61, 651.
(6) Miller, E. M , Dockerty, M. B , Wollaeger, E and Waugh, J. M. (1951) *Surg Gynec. Obstet.* 92, 172.
(7) Orr, T. G. (1945) *Surgery* 18, 144

SECTION III

OPERATIVE SURGERY

OPERATIVE SURGERY UPON THE BILIARY TRACT

Anæsthesia. The choice of anæsthetic agents is wide, provided that adequate relaxation of the upper abdomen is obtained. This is probably best achieved by the liberal use of relaxants such as curare with a basal inhalation anæsthetic. A severely damaged liver may be unduly sensitive to morphia and pentothal, while chloroform is no longer used. Annoxæmia must be avoided and cyclopropane allows of high oxygen inhalation.

Position on the Table. The patient lies fully supine upon the table. It has been the custom in the past to try and improve exposure by the use of bridges introduced under the patient's back at about the level of the twelfth rib. Objections are raised to this on the grounds that they lead to considerable post-operative pain and add materially to

operative shock. Furthermore, the aid which they give is not great and their use has consequently been abandoned by many surgeons.

It is sometimes helpful after the peritoneum has been opened to lower the table at the foot end in order that the intestines should gravitate away from the operative field and the liver descend from under the costal margin.

Incision. The paramedian, the rectus split and the Kocher incision are in common use for gall bladder surgery. In addition to these the Satinsky type of approach is sometimes of use in difficult operations upon the common duct, such as may be required in cases of stricture (*see* Fig. 77).

(i) The Paramedian Incision

The paramedian incision is usually employed when the operation is more in the nature of a laparotomy and it is not certain that gall bladder surgery will necessarily be carried out. It is made in the upper abdomen about an inch laterally to the medial border of the right rectus muscle from a point about one inch below the costal margin to a little below the umbilicus. The anterior rectus sheath is incised in the same line and the muscle reflected laterally after detachment of the tendinous intersections. The posterior rectus sheath, extraperitoneal fat and peritoneum are incised in the same line as the anterior sheath. This incision is frequently tedious to perform and moreso to close in a very obese subject. It is time consuming, but fairly secure against subsequent herniation provided that where necessary drains are brought out at a different point and the wound closed with interrupted sutures.

(ii) Rectus Split Incision

The rectus split operation carries few advantages over the former except that it can be more laterally placed and is slightly quicker to perform. It is perhaps more prone to hernia formation than the former and necessarily denervates the medial half of the split muscle. It is sometimes used for cholecystostomy.

(iii) Kocher's Incision

The Kocher's incision is perhaps the incision of choice for all gall bladder surgery. It is certainly so when a further operation is required upon a patient who has already had a paramedian incision. It is both easier and quicker to make and to close in obese subjects. It is no more prone to hernia formation than the previous and provides perhaps the best exposure when it is reasonably certain that the operation will be upon the biliary tract

It is true that some denervation of the rectus may result from division of the eighth intercostal nerve, but the denervated area is often small; and because the upper rectus is contained within rigid, fibrous compartments post-operative weakness is minimal. Kocher's incision is made parallel to and about an inch below the right costal margin from the linea alba to a variable point at about the anterior axillary line. It passes, after skin and fat, through the anterior rectus sheath and linea alba medially and across the aponeurosis and fibres of the external oblique muscle laterally. The next stage carries it through the rectus muscle itself and laterally through the internal oblique in line with its fibres. At this stage the branches of the superior epigastric artery are secured and the

eighth and perhaps ninth nerves are seen emerging from the costal margin between the internal oblique and transversus muscles. As far as is possible they are retracted laterally but division of one of these may be required. The posterior rectus sheath and the transverse abdominal muscle are next divided in the same line and the peritoneum incised.

This incision is usually closed in two layers. No attempt is made to suture the muscle itself but in the first layer the peritoneum, posterior rectus sheath, transversus abdominis and internal oblique muscles are sutured in one layer. The second layer includes the anterior rectus sheath and external oblique muscle, the drains being brought out through a separate opening. The incision is best closed with interrupted sutures.

(iv) SATINSKY INCISION (*see* Fig. 77, page 186).

This is reserved for tedious and difficult reconstructive operations on the ducts and is described under biliary stricture.

Exposure. Further measures can be carried out in order to improve the exposure. Firstly a hand is passed into the right subphrenic space in order to admit air and thereby allow the liver to move independently to the diaphragm. This manœuvre is of doubtful value, but is always worth trying when exposure is difficult. The packing off of the area is by far the most important of all measures and well repays care and time spent in this way. A hand is first inserted down to the foramen of Winslow, the duodenum and hepatic flexure of the colon are retracted caudally and a large swab inserted over the hand, which is then withdrawn. A Deaver's retractor is placed over the swab and retraction maintained towards the feet. The assistant's hand is sometimes preferable to the metal retractor. Another hand retracts the stomach towards the left and a swab placed over this and maintained with another Deaver protects the field from visceral intrusion from this direction. The liver edge is then retracted upwards. In this way the gall bladder, the under surface of the liver, the lesser omentum and its contained structures are exposed fully to view and all operative measures can thus be carried out safely under full direct vision. Before proceeding a swab is usually tucked into the foramen of Winslow and Morrison's pouch to collect any spillage which may gravitate to this area.

Cholecystostomy

This operation is used in the following circumstances.

(1) In acute cholecystitis when, owing to the condition of the patient or the severity of local inflammation, cholecystectomy is inappropriate.

(2) As a means of providing external biliary drainage when the cystic duct is patent.
- (*a*) In acute pancreatitis.
- (*b*) As a preliminary stage to the excision of obstructive pancreatic lesions.
- (*c*) In rare cases of common duct stone with a very sick patient.

(3) In cases of severe suppurative cholangitis.

(4) After removal of stones from a gall bladder not requiring excision.

Drainage of the biliary tree is best secured by a T-tube in the common duct. Cholecystostomy should only be preferred where choledochotomy is contraindictated. It is most useful where biliary tree and gall bladder require simultaneous drainage.

After the peritoneum is opened and cholecystostomy is indicated the fundus of the

gall bladder is isolated by packs and three Babcock's forceps applied in a ring about the size of a penny around the fundus. It is not always that the state of the tissues will support any forceps, in which case the fundus may be circumscribed with an atraumatic catgut stitch. A trocar and cannula attached to a sucker is then thrust through the centre of the ring and the trocar removed so that the sucker empties the gall bladder of bile and debris. The viscus itself will now be more manageable, and after withdrawal of the cannula a finger or scoop or Desjardin's forcep may be inserted to remove all stones and solid matter. At this stage it may be possible to appreciate the patency of the cystic duct if the gall bladder is seen to refill after a preliminary evacuation. When the interior of the gall bladder is as clean as possible the region of Hartmann's pouch and the cystic duct should be systematically palpated from the exterior for the presence of stones, which may be milked into the cavity of the gall bladder and removed. It is often possible to pass a probe down the cystic duct into the common duct in order to establish patency, but the necks of some gall bladders are too devious or narrowed to permit its passage.



Fig 93 Normal cholangiogram through a cholecystostomy tube

It must be emphasized that if the function of the cholecystostomy is to drain the biliary tract the patency of the cystic duct must be established beyond doubt and if this proves impossible then a T-tube in the common duct is a safer measure. When, however, the cholecystostomy aims to do no more than decompress an acutely inflamed gall bladder the patency of the cystic duct is not so important, although it is still desirable.

When the surgeon is satisfied that the biliary passage is clear of all stones a soft rubber drainage tube about the size of the little finger is inserted for a distance of about two inches through the incision in the fundus. A purse-string suture is now inserted into the gall bladder wall around the incision and finally through the wall of the tube itself. This is then tied and at the same time the fundus is invaginated by the slight advancement of the tube In the same way a second, and perhaps a third, purse-string is inserted so that the fundus comes to be turned in like a safety inkwell. It is a common mistake to insert the tube too far in the first instance so that after invagination it is impacted into the neck of the gall bladder and there obstructs free drainage and presses hard upon the wall. The issuing limb of the tube is now passed through the greater omentum, which helps to form a seal around it between gall bladder and parietal peritoneum. The tube should emerge through a conveniently placed stab incision. In this way the original abdominal incision can be sewn up in its entirety and the chances of subsequent sepsis

and herniation thereby reduced. Before closing the wound the gall bladder wall next to the tube is secured to parietal peritoneum by two or three catgut stitches in order to minimize intraperitoneal leakage and shorten the subsequent track.

Management of the Tube. In acute cholecystitis the tube is retained while the signs of inflammation persist after which time it may be removed, although usually not less than a week after its insertion. It can be withdrawn without difficulty at this time for the catgut will have been destroyed and the track perfectly sealed. In a normal case the biliary discharge may persist for two or three days, but the fistula should then gradually close. If the cystic duct remains impervious a mucus fistula results, this being the secretion of the gall bladder mucosa. Should there be any distal obstruction of the common duct then a biliary fistula will often persist until such obstruction is overcome. If in such a case healing should occur despite obstruction then jaundice will return, though an intermittent obstruction such as a stone may give intermittent jaundice. Cholangiography may be carried out through the cholecystostomy tube and is often very informative (Fig. 93).

Cholecystectomy

The steps in the technique of cholecystectomy do not differ according to whether an acutely or a chronically inflamed gall bladder is being removed, although the ease with which they are performed varies considerably. In the acutely inflamed gall bladder much will depend upon whether the present attack is superimposed upon a chronically inflamed viscus, or whether prior to the attack the gall bladder had been relatively normal. Difficulties arise as a result of fibrous tissue being laid down across and between those anatomical planes and structures upon which the surgeon relies during cholecystectomy. When the structures in the lesser omentum are matted by fibrous tissue and overlain by an enlarged adherent cholecystic gland they may be so difficult to identify that serious accidents occur.

Routine cholecystectomy usually commences at the neck and after detachment of any adhesions between duodenum or colon and the gall bladder, an incision is made in the peritoneum overlying the region of the cystic and common bile ducts, passing down the right anterior aspect of the free border of the lesser omentum. These folds are now reflected to expose the junction of the two ducts.

It is a vital maxim that no structure should be divided until all structures, that is to say the common bile duct, the common hepatic duct, the cystic duct, the cystic artery, and vein and the right hepatic artery, have been fully visualized and displayed. The anatomical anomalies in this area are important and numerous and the only safe procedure is to dissect the region as though the individual were a new species never before examined as no amount of foreknowledge of anatomical likelihoods will inform the surgeon of the precise arrangement in the individual case. The most accessible structure is usually the cystic duct and where it is tortuous a Moynihan's forcep may be applied to Hartmann's pouch in order by traction to straighten it out. After identification and demonstration of its termination by the direct display of the common and common hepatic ducts, attention is now paid to the triangle bounded by the liver above, the cystic duct below and the common hepatic duct to the left (Fig. 93). It is in this area that the cystic usually leaves the right hepatic artery and both these structures must be identified where possible. It is extremely easy to clamp or divide the right hepatic artery in this

situation, more especially as the cystic artery under traction appears to be the direct continuation of what is actually the proximal part of the right hepatic; the distal part coming off at an angle on the deep surface and being hidden. This accident will be avoided if the cystic artery is secured as a routine well up on the gall bladder wall.

All these structures now being discovered and doubts as to their identity dispelled, *division of the cystic duct may be proceeded with. It is better accomplished with*

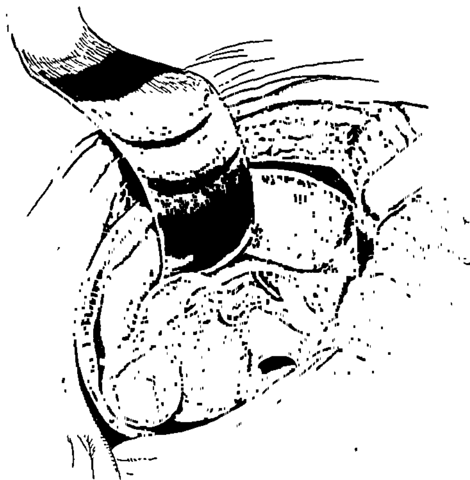


FIG 94 The peritoneum covering cystic duct and its termination has been opened displaying the common bile duct, common hepatic duct, cystic artery and its origin, in this case, from the right hepatic. These structures are lying for the most part in the triangle made by the cystic and common hepatic ducts and the *inferior surface of the liver*

aneurysm needles than by a clamp, which is open to the danger of including part of the common duct and other structures which are not intended. The ligature nearest to the common duct is tied so that the duct is not narrowed, and no blind diverticulum remains.

After division of the cystic duct exposure of the cystic artery is often made more easy. It is under-run with aneurysm needles and divided between ligatures, the vein and artery being taken together in most cases.

Traction is now placed upon the region of Hartmann's pouch, another forcep (Duval) applied to the fundus and the gall bladder held at full length and retracted away from the liver so that the peritoneal folds on either side connecting the gall bladder to the liver are under tension. They are then incised in such a way as to leave a short flap

at either margin, about 1 cm. in breadth, with which at a later stage to cover the gall bladder bed. This incision is carried down to such a depth as will allow of the reflection of these flaps. A finger is then inserted from below into the space between gall bladder and liver and a plane developed between these two structures, which in favourable cases can be carried up to the fundus. By the extension of this space the gall bladder is lifted clear of the peritoneal flaps until only the fundus remains attached. It is often useful to utilize this latter attachment as a retractor to hold up the gall bladder bed while attention is paid to hæmostasis and the suture of the flaps covering the raw area. This

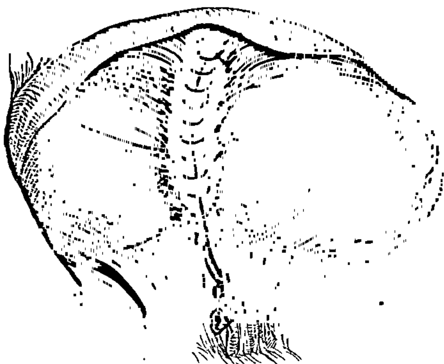


FIG 95. The gall bladder bed has been sutured to control hæmorrhage and minimize biliary leakage The free border of the lesser omentum is better left open The cystic duct stump is ligated close to the main duct

suture usually starts at the point on the lesser omentum where the original incision was made and is carried continuously up to the region of the fundus (Fig. 95). The last remaining attachment can now be severed and the gall bladder is free.

A corrugated drain of about an inch in width is passed down to the foramen of Winslow. This is a vital part of the operation as some biliary leakage is to be anticipated in nearly all cases. This bile is said to come from (i) minute biliary canaliculi, which may be passing from liver to gall bladder, (ii) from the division of accessory ducts of varying importance, which have escaped notice; (iii) from the unexpected injury to or division of normal ducts. Failure to drain these cases will result either in a generalized biliary peritonitis or a localized collection of bile which may eventually find its way through the wound, or persist and become infected, so accounting for some cases of unexplained post-operative jaundice, from its slow absorption. The drain is usually retained until the fourth post-operative day, in the absence of an abnormal biliary leak.

Retrograde Cholecystectomy. Some surgeons prefer to commence a cholecystectomy

at the fundus, working towards the cystic duct. The disadvantages of this method lie in the fact that the dissection is being carried out prior to the control of the cystic vessels and is on this account more hæmorrhagic. Nevertheless, this method is a useful one to hold in readiness for those cases in which the anatomy in the lesser omentum is so obscured or matted as to defy an adequate dissection.

In these cases, with care and by following close to the gall bladder wall where it is separated from the liver, the surgeon is naturally led to the junction of cystic and common

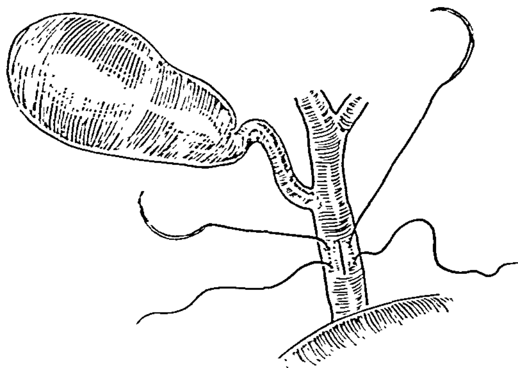


Fig. 96. Opening the common duct: placement of guy sutures.

hepatic ducts. In this method the cystic artery is encountered on the way down, but provided that the dissection is everywhere as close as possible to the gall bladder wall itself damage is unlikely to result.

Exploration of the Common Duct

The decision will have to be made in each case as to whether the common duct needs to be explored. The various indications have been outlined in the previous section, but a decision is probably better made prior to cholecystectomy, after a thorough palpation of the region, for the following reasons:

(1) Sometimes the condition of the lower common duct may influence the surgeon against cholecystectomy.

(2) Once the packs are in place it is no longer possible, without removing them, to palpate the entire duct.

(3) Prior to cholecystectomy the operative field is still clean (except in the acute case) and intraperitoneal manœuvres are better carried out at this stage.

Before the duct is actually opened it is as well to have the cystic duct under control.

if only temporarily, since at any stage during the exploration some stones may pass down the cystic duct into the part already pronounced clear of them.

The duct is then opened in the following manner:

The antero-lateral surface of the supraduodenal portion of the common duct is fully cleared below the normal entry of the cystic duct. Two stay sutures are then

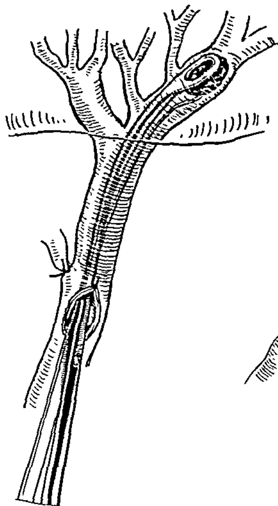


Fig 97 An intra-hepatic stone is reached with Maingot's forceps.

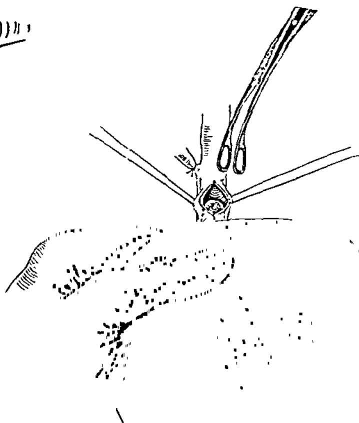


Fig 98 A common duct stone is milked up from the retropancreatic region and removed through the choledochotomy incision with Desjardin's forceps.

inserted longitudinally about ½ cm. apart. A longitudinal incision is then made between them for a distance of about 1½ cm. (Fig. 95). If there is uncertainty as to the identity of the common duct a hypodermic needle attached to an aspirating syringe may be inserted and the contents withdrawn for inspection. Its appearance may provide another indication for opening the duct.

The upper reaches of the biliary tract are first explored with a malleable scoop (which can be bent to an appropriate angle to pass up the common and into either *hepatic* duct), or with appropriately shaped forceps (Maingot) (Fig. 97). Any stones which are encountered are withdrawn. The lower reaches are now probed in the same manner. If a stone is palpated it can very frequently be milked up to the region of the incision and there

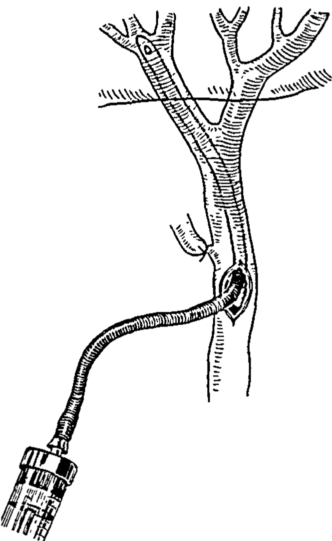


Fig. 99 Syringing the higher reaches of the intrahepatic biliary tree in the hope of washing down any high-lying stone

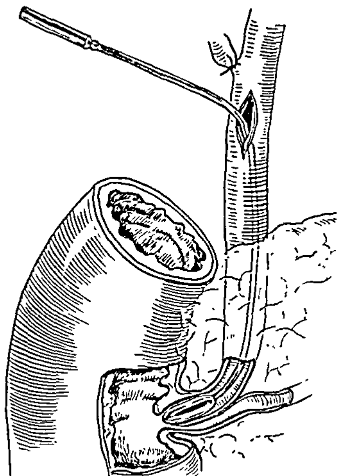


Fig 100. The passage of Bakes' dilators through the sphincter of Oddi

withdrawn with Desjardin's forceps (Fig. 98). Dependence cannot be placed upon the sounding of a stone as they are sometimes so soft as to make no sensation of contact. When no further results are obtained from scooping, the patency of the papilla should be tested with the probe. By bending it to a suitable shape so that the lateral deviation of the lower part of the duct is accommodated it can be manœuvred through the papilla and into the duodenum. Its sudden negotiation of the final opening is usually distinctly felt, but in

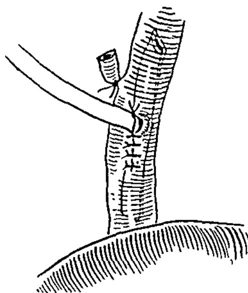


FIG. 101 The common duct wound closed with T-tube *in situ* and one suture passing round the neck of the T, but not obstructing it

any case the presence of the probe within the second part of the duodenum can be verified by palpating it as it moves freely within. When the probe is *in situ* the duct itself can be further palpated as its exact anatomical position is now easily ascertained and any dubious nodules in the posterior part of the pancreas investigated. It is possible for the probe to enter the duodenum bypassing a small stone and this should be detected by palpation at this stage.

The ducts are finally washed by the use of a fine rubber urethral catheter attached to a syringe. This is passed as high as possible into the hepatic region and 20 cc. of saline forcibly injected (Fig. 99). In this way small pieces of gravel are washed through the duct incision. This is now repeated for the lower reaches and the procedure concluded by the dilatation of the duct papilla with special graduated bougies (Bakes' or Bochus') up to a diameter of 8 or 9 mm (Fig 100). This would allow any fine particulate matter which remains to pass into the duodenum with the flow of bile. A T-tube is now selected of a pattern in which the indwelling portion consists of only half the circumference as this causes less obstruction within the duct and is more easily withdrawn when the time comes It is placed within the duct by first inserting the upper end and part of the horizontal limb into the upper part of the duct The lower limb is now inserted and the horizontal limb partially withdrawn so that the indwelling portion passes downwards into the correct position. The incision in the duct is now closed at the inferior margin by interrupted fine catgut so that the issuing limb fits snugly into the upper part of the incision, the remainder being closed in as watertight a manner as possible. The stitch next to the issuing limb, after being tied, encircles the tube itself to hold it in place (Fig 101).

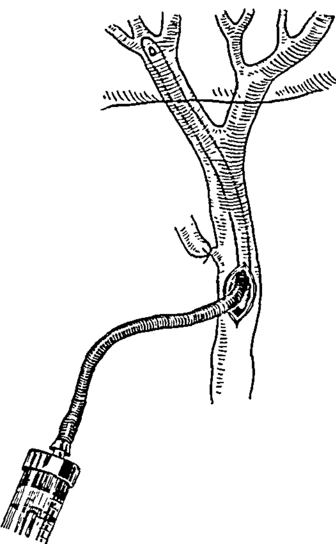


FIG. 99. Syringing the higher reaches of the intrahepatic biliary tree in the hope of washing down any high-lying stone

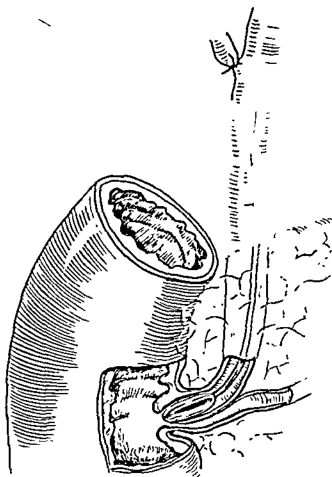


FIG 100 The passage of Bakes' dilators through the sphincter of Oddi.

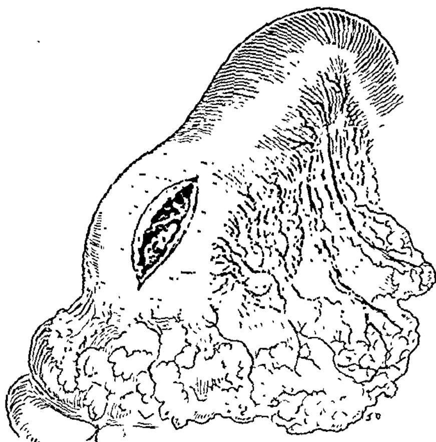


FIG 104 Transduodenal approach to the papilla.

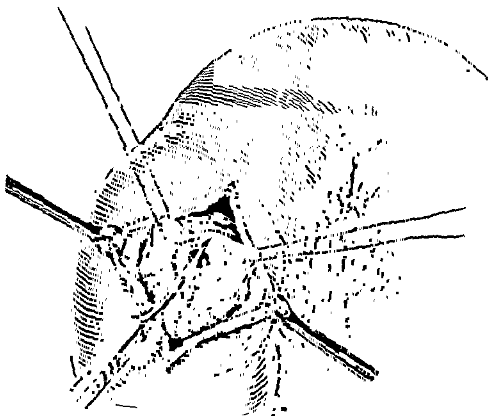


FIG 105 Raising of the papilla by *guy sutures*, through the anterior duodenal wall The sphincter is being divided at its superior margin for an impacted low-lying stone. Transduodenal papillary choledochotomy

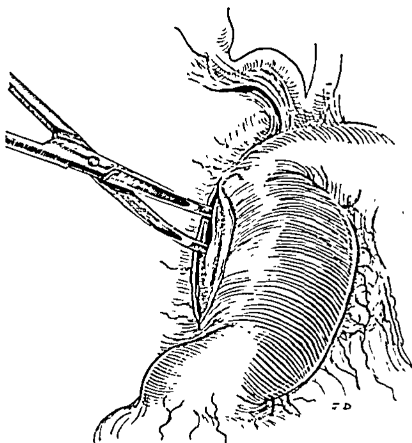


Fig. 102. Mobilizing the second part of the duodenum The lateral peritoneal reflection has been incised and the retro-duodenal plane is being developed.

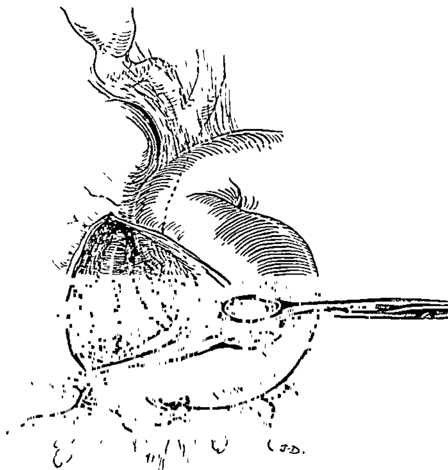


Fig. 103. A further stage of mobilization of the duodenum. The posterior surface of the pancreas and common bile duct are visible and the mobilization can be extended as far as the inferior vena cava

the posterior duodenal wall and the anterior wall of the duct (Fig. 107). The stone is delivered through the incision, after which duct and duodenum are anastomosed by suturing the incised edges of their walls. This is a form of transduodenal choledocho-duodenostomy and is usually described under Kocher's name. Such methods are probably less likely to lead to troublesome biliary fistulæ than the extraperitoneal choledochotomy which provides an alternative access to the low impacted stone. The duodenal incision is sewn up transversely (Fig. 108).

Once the duct is opened the surgeon must be certain of a clear passage into the duodenum and in difficult cases the probe can only be passed or failure to pass it explained by opening the duodenum. The cause of obstruction may then be ascertained, whether it be stone or stricture or papillary fibrosis or new growth, and appropriate action taken. A failed probing of the papilla is likely to produce a post-operative obstruction and consequently, in the absence of new growth, the papilla, when this is obstructing the probe, should be divided at its upper margin (papillary meatotomy) and a long arm T-tube inserted through it.

It has been emphasized by some surgeons that in difficult procedures upon the common duct, structures are more easily handled with the surgeon standing on the patient's left-hand side. In this way the duct may be more naturally held in the surgeon's left hand while the right hand is free for further action.

Post-operative Care

(i) Chemotherapy

In infected cases, where there has been a pre-operative pyrexia, or the bile at operation is turbid, or dissemination of infection has taken place at operation, the use of post-operative chemotherapy is advisable. For this purpose, in view of the frequency of intestinal organisms, streptomycin or aureomycin are the drugs of choice, although the sulphonamides are almost as effective where there is no vomiting and they can be retained.

(ii) Sedation

It is usually taught that morphia augments contraction of the sphincter of Oddi and is responsible for post-operative discomfort and perhaps vomiting following operations upon the biliary tract and many surgeons will not permit its use on this account. Other sedatives such as tracentin or pethedine are said to be less objectionable in this respect.

(iii) Drains

The discharge around the drain is carefully noted and whereas it is normal to expect biliary staining of the dressings to occur up to the third post-operative day if it should be copious or persist much after this time it may indicate that there has been damage to the main ducts or that biliary obstruction remains. The drain is normally removed at about the fourth or fifth day, but in the presence of such a discharge it should be retained for about a week.

(iv) T-tube

From about the sixth post-operative day, if the bile is clean, the tube can be closed so that all bile now passes into the duodenum. If this is unaccompanied by untoward symptoms the tube may remain closed, but if pain results or leakage of bile around the tube the spiggott should be removed and the procedure repeated upon the following day.

At this stage the patency of the tube is tested as every now and again an impervious one is supplied. The tube is brought out through a separate incision in the abdominal wall along with the corrugated drain. It is stitched to the skin to prevent accidental withdrawal by patient or nurse.

Additional Manœuvres

(i) MOBILIZING THE DUODENUM

When palpation and manipulation of the lower common duct is difficult mobilization of the second part of the duodenum may be carried out as described by Moynihan and Kocher. This involves the incision of the posterior parietal peritoneum along a line parallel with and about half an inch from the lateral border of the second part of the duodenum (Fig. 102) Through this incision a plane is developed between the duodenum and pancreas anteriorly and the perirenal fat and kidney and its vessels posteriorly. This not only enables a bidigital grip to be obtained on the common duct, but allows the pancreas and duodenum to be reflected and rotated as far as the mid-line so that the posterior aspect of the duct and pancreas may be inspected (Fig. 103). In this way the duct can be followed right into the duodenum, although it is necessary in some cases to incise a thin pancreatic covering

(ii) OPENING THE DUODENUM

When the stone is impacted at or immediately above the papilla and cannot be milked into the duodenum or up into the supraduodenal portion of the duct, or persistent efforts fail to secure the passage of the probe through the papilla, there should be no hesitation in opening the anterior wall of the second part of the duodenum in a longi-

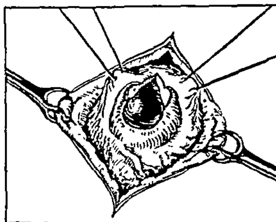


FIG 106 The sphincter divided, the stone presents and can now be lifted from the wound

tudinal direction (Fig. 104) In this way the papilla may be approached across the cavity of the duodenum (Fig. 105) and by a meatotomy the stone can be delivered into the duodenum (transduodenal papillary choledochotomy—McBurney) (Fig. 106). If the stone is not actually visible at the ampulla but is impacted in that portion of the duct immediately above, an incision may be made from the lumen of the duodenum through

If discomfort invariably follows the closure then obstruction at the distal end of the duct is probably present.

The tube is normally retained for a period of 12–16 days, at the end of which time it may be withdrawn by a steady pull, the stitches having been absorbed. Prior to its removal a routine cholangiogram with a water soluble medium should be carried out in order to visualize the biliary tree (*see* previous section).

The tube is retained longer than the usual time in the following circumstances:

(1) When pain or biliary leakage persistently follows the closing of it.

(2) When the bile is still turbid and infected.

(3) When the cholangiogram shows suspicious filling defects or persistent dilatation of the biliary tract (hydrohepatosis), or the medium fails to enter the duodenum.

(4) When a long arm or ordinary T-tube is used as a splint in cases of stricture.

In the absence of complications the patient usually leaves his bed for the first time on the second post-operative day, except in cases of acute cholecystitis when it is advisable to await the subsidence of the local peritonitis. Early rising is believed to discourage chest complications and venous thrombosis.

Complications

(i) Hæmorrhage

Hæmorrhage in the region of the gall bladder neck sometimes results when the cystic artery escapes or may follow injury to other neighbouring arteries (especially when anomalous) and is particularly dangerous because the surgeon is tempted to grab with the hæmostat in the vicinity of the vital ducts and arteries. It is better in such circumstances to grasp the hepatic artery between finger and thumb by inserting the forefinger through the foramen of Winslow and pressing the thumb down upon it. In this way the blood may be sucked away and momentary release of pressure reveals the bleeding point, which can then be dealt with under direct vision. Serious post-operative hæmorrhage occasionally occurs as a result of insecure ligation of the cystic artery. It must be remembered that all jaundiced cases are particularly prone to bleeding and for this reason should be adequately prepared with vitamin K prior to operation.

(ii) Sepsis

Most gall bladders are removed because of inflammation, acute or chronic, and the wound is consequently often contaminated with organisms at the time of operation. The patients are obese and the presence of bile and drains further add to the risk of infection. Wound sepsis is therefore not uncommon following this operation. It should be anticipated by the use of antibiotics, by the gentle handling of the tissues and the provision of adequate wound drainage. For this reason the wounds are better sewn up by interrupted chromic catgut throughout as non-absorbable suture material may result in sinus formation. What appears to be a wound abscess may, when it bursts or at incision, often prove to be a collection of bile loculated beneath the liver surface. Sepsis predisposes to dehiscence or eventual incisional hernia.

(iii) Damage to Hepatic Arteries

Ligature of the common hepatic artery or of the right hepatic artery was always said to be fatal and no doubt on many occasions it is so. This opinion may have to be

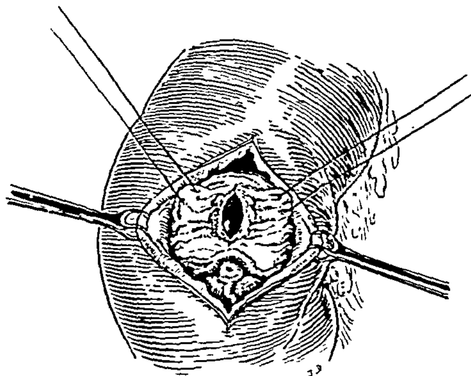


FIG. 107 The stone impacted slightly above the papilla requires a choledochotomy through the posterior duodenal wall leaving the papilla intact. Duct and duodenum are united around the margin of the incision by interrupted sutures.

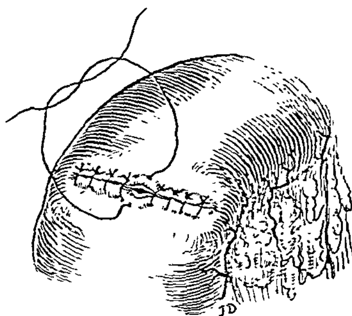


FIG. 108 The longitudinal duodenal incision is sewn up transversely.

The normal course when presented with such a complication is to try to secure adequate drainage when a T-tube exists and to combat biliary tree infection with appropriate antibiotics. Such an expectant attitude is justified and usually rewarded by a gradual diminution of the jaundice. In the face of a gradually increasing icterus after two to three weeks of conservative treatment re-exploration will be required. The abdomen is re-opened and if a large collection of bile is found it should be drained and its source discovered if possible. The common duct is sought and if intact re-explored and a free passage insured. If a stricture or occlusion is found it should be dealt with according to the principles previously considered.

Late jaundice occurring several months or years after cholecystectomy may be due to:

(1) Common duct stone.
(2) Stricture of the common duct.
(3) Pancreatitis.
(4) Cholangitis due to duct deformity or inflammation of a cystic duct remnant.
(5) Biliary cirrhosis.

(vii) Duodenal Fistula

This is a serious complication and may lead to the loss of a patient through failure to correct the resulting dehydration and biochemical derangement.

It may be due to:

(1) Operative trauma to the duodenum.
(2) The pressure of a drain which is too stiff and too long retained.
(3) Faulty closure of a deliberate duodenal incision.
(4) Severe implication of the duodenum in the pericholecystic inflammation.

The skin must be protected from the digestive juices by means of barrier ointments and continuous suction at the fistula and nasogastrically. The fluid, chloride, sodium, and potassium loss must be made good by intravenous therapy, and feeding carried out by the same route with invert sugars and protein hydrolysates. Under this regime most fistulæ will heal spontaneously, though a jejunostomy may be required if, after a fortnight, the fistula still remains large. It has occasionally proved possible to pass a Miller-Abbott or Harris tube to the upper jejunum for feeding purposes and thus avoid the jejunostomy.

(viii) Hepatic Failure and Liver Deaths

The loss of a patient from liver failure is a possibility in any operation upon the biliary tract but its likelihood is greatly increased in the presence of:

(1) Deep jaundice.
(2) Cholangitis.
(3) White bile in the common ducts.
(4) Pre-existing cirrhosis.

The understanding of this mode of death is far from complete. The greatest risk is in the first post-operative week when the patient may gradually lapse into a deepening coma. There may be a preliminary phase of excitement and signs of widespread central nervous disorder. The cause of the coma is variously but uncertainly ascribed to electrolyte disturbance, vitamin deficiency, nitrogen retention or "toxæmia." The depth of coma is not proportional to the degree of jaundice, nor is it clear exactly what is happening to the liver cell which brings about the failure, which is to a certain extent

slightly modified in view of the more recent modern knowledge, and the therapeutic ligature of hepatic arteries, though at a different site and in a different disease. The closer to the liver that the ligature is placed the more dangerous it is. The result may be a diffuse anærobic infection which is rapidly fatal, but which may be prevented, at all events experimentally, by the prophylactic use of antibiotics and sulphonamides.

(iv) Injury to the Bile Ducts

(1) Ligature of the common hepatic duct.

This rapidly results in a severe post-operative jaundice. No biliary fistula occurs unless the duct subsequently gives way at the point of ligature.

(2) Incision of the common bile ducts.

If, unknown to the surgeon, the common bile duct is incised a biliary leak with a variable degree of biliary peritonitis will ensue, resulting in a post-operative fistula which, provided that there is no distal obstruction to the common duct, should heal of its own accord within a week or two following operation.

(3) Crushing of the bile ducts.

The bile ducts may be accidentally crushed by an artery forcep. This may result in the duct wall giving way during the first post-operative week, in which case a temporary fistula may result. The accident may lead to a stenosing condition of the duct with a consequent late onset of obstructive jaundice, perhaps six months to several years post-operatively. A mechanical narrowing of the duct may lead to late intermittent infective obstructive jaundice.

(v) Retained Common Duct Stones

If the common duct is unexplored, or stones are overlooked, their continued presence may result in early post-operative jaundice or persistence of dyspepsia and pain as before operation; or when the duct has been opened a persistent biliary fistula may refuse to heal.

Post-operative biliary fistula may thus be due to:

(1) Severance or injury to normal or accessory ducts.

(2) Distal biliary obstruction due to stone or stricture or pancreatic disease.

(3) Operative closure of the main ducts.

(vi) Post-operative Jaundice

Immediate post-operative jaundice is rarely due to hæmolysis of an incidental transfusion, but more often can be ascribed to.

(1) Cholangitis and hepatitis

This sometimes follows operative handling of the acutely inflamed gall bladder or manipulation of the common duct which is already inflamed. It is accompanied by a high temperature.

(2) Obstruction to the common duct due to a ligature, a retained stone, or a blocked T-tube.

(3) Absorption from a loculated subhepatic intraperitoneal collection of bile which has failed to drain onto the surface.

(4) Aggravation by handling of pre-existing pancreatic pathology.

(5) Biliary cirrhosis.

common bile duct receives severe manipulation and full post-operative biliary drainage is not provided. It is against such risks that the routine use of post-operative antibiotics is directed.

Recurring attacks of cholangitis may follow cholecystectomy by some months or years and suggest a narrowing of the main duct or a common duct stone or rarely recurrent infection of a persistent cystic stump. Jaundice may or may not be associated. Such a history, if persistent, requires an investigation of the state of the common duct by cholangiography and perhaps even choledochotomy.

(xi) Post-cholecystectomy Syndromes

It is not unusual for patients to continue to complain following removal of the gall bladder of symptoms not unlike those experienced pre-operatively. As in the excision of many other organs the most grateful patients are to be found amongst those who suffered most before surgical treatment. As previously stated the results of cholecystectomy are most disappointing when undertaken upon flimsy indications or for non-calculous gall bladder disease. They are still less satisfactory when the symptoms are coming from other viscera. Persistent symptoms are sometimes due to a neglected common duct stone, a stricture, a chronic pancreatitis, a duodenal ulcer or a hiatus hernia; in fact a mistaken diagnosis.

Apart from such instances there are undoubted cases in which symptoms appear to arise in the remaining biliary tract and are sometimes referred to as biliary dyskinesia.

Following cholecystectomy the extrahepatic ducts usually dilate to almost twice their normal size and this is also found when the gall bladder functions are destroyed by disease. The normal flow of bile from the liver is continuous whereas its discharge into the duodenum is intermittent and largely dependent upon feeding. These arrangements are normally made compatible by the interposition of the gall bladder which by providing storage space and abstracting water prevents a rising pressure in the ducts. Following cholecystectomy it seems that the sphincter of Oddi is loth to learn new habits and continues to hold back the bile against the secretory hepatic pressure so that mild intermittent biliary obstruction exists This mechanism has been held to account for:

(1) The dilated ducts after cholecystectomy.

(2) The occasional dilatation of a persistent cystic stump.

(3) The post-cholecystectomy syndrome.

(4) The necessity for T-tube drainage following choledochotomy and cholecystectomy

(5) Post-cholecystectomy pancreatitis perhaps due to biliary reflux.

(6) The occasionally elevated blood cholesterol and alkaline phosphatase after cholecystectomy.

(7) The increased susceptibility to cholangitis.

Such symptoms will be less noticeable to the patient whose diseased gall bladder could no longer expand or concentrate before operation. There is a feeling that dilatation of the sphincter at the time of choledochotomy may discourage the later onset of such symptoms.

When they do occur, however, the diagnosis should be reappraised and if confirmed all conservative methods tried. These include cholagogues, antispasmodics, dietary experiments, etc.

reversible either spontaneously or sometimes by treatment. The situation may be analogous to the anuria of sudden urinary decompression or may follow post-operative fulmination of a pre-existing cholangitis. Sometimes there is anatomical interference with the hepatic arteries and main bile ducts or an unrecognized internal hæmorrhage: these cases also pass as "liver deaths."

There is a variant of the usual cholæmic picture in which the kidneys seem to fail at the same time as the liver. In these patients there is anuria or oliguria with a mounting blood urea and the terminal picture is a mixture of cholæmia and uræmia. The mechanism of this type of termination has never been adequately explained.

Modern opinion tends to question the unexplained "liver death" believing that in most fatal cases an adequate cause can be found in the form of intrahepatic inflammation, suppuration, cellular necrosis, infarction or hæmorrhage. Cirrhosis in itself, though often present, does not necessarily account for the fatal issue. The syndrome is not confined to liver cases.

The treatment is chiefly prophylactic and consists of the protection of the liver cell with an adequate store of glycogen, the pre-operative provision of vitamins when lacking, the control of biliary infection with antibiotics, careful handling of the hepatic vascular bundle and perhaps most important the selection of an operative procedure within the limits of tolerance of the individual patient.

When coma or cholæmia threatens post-operatively glucose and intravenous fluid should be provided, though overloading is a real danger if the kidneys are inefficient. Vitamin B is usually given intravenously. Other forms of intravenous therapy such as liver extract, choline and methionine, albumen and heparin are advocated from time to time. The most recent addition to this questionable armamentarium is the use of intravenous glutamic acid.

(ix) Recurrent Pancreatitis

Attacks of pancreatitis may have preceded the biliary tract surgery which is sometimes undertaken on this account, to remove a diseased gall bladder or common duct stones. Such measures are not always curative and in some cases the pancreatitis occurs for the first time in the immediate post-operative period, possibly as a result of trauma or post-operative obstruction at the sphincter of Oddi. Such an occurrence is difficult to diagnose in the post-operative abdomen but accounts for a few deaths and several instances of inexplicably stormy convalescence. Most cases pass unrecognized but when suspected can sometimes be confirmed by a serum amylase. There is little to be done other than the relief of pain, attention to fluid and electrolyte needs and allowing free biliary drainage where a T-tube already exists.

Pancreatitis may arise at a much later date and may indicate a recurrent or overlooked common duct stone. In the chronic form there may be obstructive jaundice requiring further surgery, and it is on such occasions that the surgeon may wish the gall bladder was still *in situ* and available for the formation of a biliary by-pass.

(x) Recurring Cholangitis

Cholangitis may flare during the immediate post-operative period and lead to multiple cholangitic abscesses throughout the liver or a more rapid liver failure in fulminating infections. This danger is most acute when an already infected obstructed

Partial Resection of the Liver
(viz. Section I, p. 139—"Tumours of the Liver")

The difficulties of liver resection are chiefly concerned with hæmostasis, the minimizing of a biliary leak and the leaving of sufficient liver tissue provided with adequate portal, hepatic and biliary channels to allow it to survive and function.

Certain parts of the liver are more suitable for resection than others. The factors which favour resection are:

(i) A pedunculated hepatic mass.
(ii) A resection from the liver phlange.
(iii) A resection of the entire left lobe.

Blood loss can be controlled in several ways.

(1) By the use of clamps applied to the liver phlange prior to the resection of a wedge. The major vessels can then be visualized on the cut surface and underrun with a stitch.

(2) By the placing of screens of mattress sutures demonstrating the wedge of liver to be excised (Fig. 109). These can be prevented from cutting through the liver by the use of thick catgut and the interposition of free omental or fascial grafts between the loop and the liver capsule (Fig. 110). The mattressed raw surfaces can sometimes be approximated by all-inclusive sutures, or the raw area covered by an omental flap (Fig. 111).



FIG. 110 The method of including a piece of muscle or omentum to prevent the stitches cutting into the liver substance

(3) By the use of the diathermy cutting knife and later coagulation.

(4) By gently clamping the lesser omentum at the porta hepatis and occluding the hepatic artery and portal vein. There is a limit to the length of time for which this can be done as the splanchnic bed becomes congested and on release of the clamp shock may develop (perhaps from the accumulation of toxic products in the stagnant blood). Portal hypertensives are more tolerant of temporary portal venous occlusion, but in normal individuals the clamp should be released every five minutes or so until the bleeding liver surface is controlled.

Very occasionally distress may require further laparotomy. If an organic explanation is still lacking dilatation of the sphincter with prolonged T-tube drainage has been advocated. This can be combined with transduodenal sphincterotomy. The

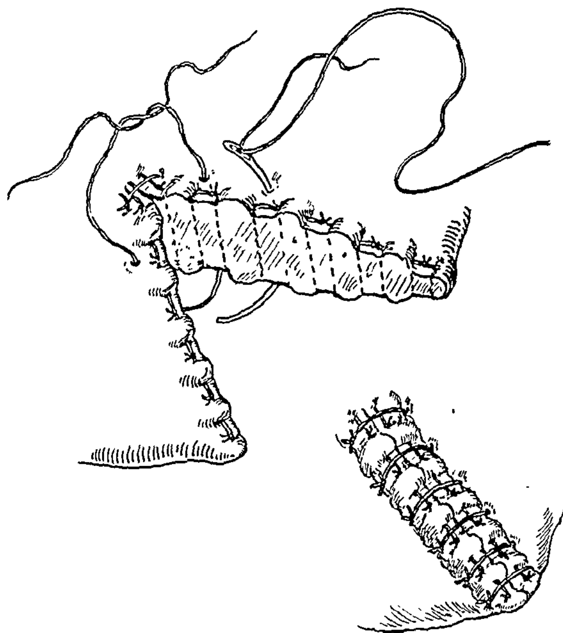


FIG 109 Wedge resection of liver Hæmorrhage from the cut edge is controlled by banks of mattress sutures which can be inserted prior to the excision The cut surface can sometimes be approximated. The classical method A blunt Kouznetzov needle is shown

T-tube used in such cases has a distal limb long enough to extend well into the second part of the duodenum and should be retained until the sphincter wound is healed and past all danger of further contracture—a period of several weeks. Other measures such as periductal stripping or vagotomy or sympathectomy also have their advocates.

(5) By the use of oxycel and similar coagulant devices interposed between the approximated raw liver surfaces.

(6) By the use of a special blunt needle (Kouznetzov) (Fig. 112), similar to a brain needle, which pushes the intrahepatic structures aside rather than transfixing them. Similarly a blunt dissector blade will cut the liver substance but spare the vessels which can be clamped as they are uncovered.

(7) By the securing and ligature, where possible, of the left hepatic artery, portal vein, and hepatic duct after bifurcation prior to the resection of the left lobe.

No liver resection should be carried out without a full antibiotic coverage in view of the probability of leaving some partly ischæmic liver tissue and the danger of clostridial infection.

A drain should always be left down to the liver area as some biliary leakage is almost inevitable.

Liver Biopsy. A representative portion of the liver edge is chosen and a doubled suture inserted and tied each side of the intended wedge excision. The enclosed tissue is then cut away without hæmorrhage.

Biliary Intestinal Anastomosis

This subject has been already dealt with in part in Section II, page 189—"Principles of Reconstruction."

The making of a new opening is required when·

(1) The existing channels are narrowed or closed:

(*a*) Due to stricture or atresia.

(*b*) Due to carcinoma of (i) pancreas.
(ii) ampulla.
(iii) duodenum.
(iv) lower common bile duct.

(*c*) Due to pressure by enlarged glands or deposits.

(*d*) Due to chronic pancreatitis.

(2) The existing channels are to be excised in the removal of a carcinoma of pancreatico-duodenal unit.

A wide choice exists between the structures selected for the anastomosis. On the one hand the gall bladder, common hepatic or bile ducts and on the other the stomach, duodenum or jejunum may be available. The choice of the biliary structures is often dictated by the height of the lesion which makes the operation necessary. Should it extend above the junction of cystic and common hepatic duct then the latter must be used for the anastomosis. When this junction is unaffected then the common bile duct or gall bladder are available and a choice must be made. The gall bladder itself when comparatively uninflamed comes easily to hand and especially after some mobilization of the fundus can be anastomosed with ease and therefore safety to almost any part of the alimentary tract, and for this reason alone it is the method usually chosen. The result is not always perfect, however, and trouble may follow.

(1) The anastomosis may completely heal, especially when the normal channels are merely stenosed and will transmit bile in varying quantities.

(2) When the common bile duct is obstructed at its lower end the distal cul-de-sac becomes a trap for all particulate biliary matter, so that during the course of months



FIG 111. A raw surface after excision may be covered by an omental flap

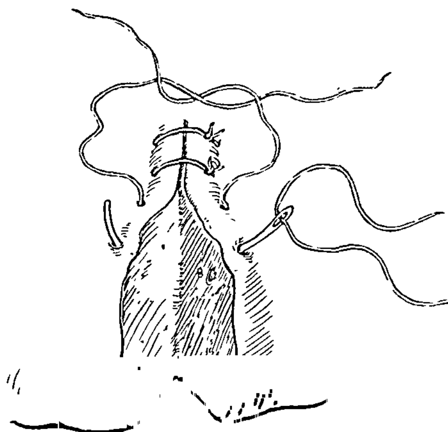


FIG 112 Straight forward closure of a wedge

months. The effect of deflection of bile so far from the gastric acid, and the risk of duodenal ulcer, particularly when pancreatic secretion is also deficient, has not yet been fully assessed.

References

(1) Boyce, F. F., Veal, J. R. and McFettridge, E. M. (1936) *Surg. Gynec. Obstet.* 63, 43.
(2) Glenn, F., and Hays, D. M. (1952) *Surg. Gynec. Obstet.* 94, 283.
(3) Heuer, G. J. (1934) *Ann. Surg.* 99, 881.
(4) Heyd, C. G. (1940) *Ann. Surg.* 111, 820.

or years it becomes completely silted up with biliary mud and sludge. In this stagnant sump organisms lurk and give rise to recurring cholangitis which is usually blamed onto intestinal reflux at the stoma. The cholangitis and liver infection may be serious enough to demand urgent operative intervention.

The frequency of this complication indicates that where the patient may live for several years, a sounder design of anastomosis is preferable. The direct union of common bile duct and intestine should be made. Indeed, were it not for the greater difficulty of this operation and the greater risk of post-operative stomal stricture it would always be the operation of choice.

(3) When the pancreas and lower bile duct are resected and the blind common bile duct stump ligated there is a tendency for this ligature to blow off or the neighbouring wall to necrose resulting in a major biliary leak. This is especially likely when pancreatic juice is also free in the neighbourhood. For this reason the common duct is preferred for the reconstitution of biliary drainage into the alimentary canal.

In former days the stomach was used for the anastomosis. It is seldom chosen now because of:

(i) The disparity in the thickness of its walls compared with the gall bladder.

(ii) The greater pressures within it as a result of muscular contraction, increasing the tendency to reflux.

(iii) The variable position of the viscus before and after meals.

The duodenum has not the same disadvantage and an anastomosis here puts the bile where it naturally should be. The duodenum is not quite as mobile as it might be, though this can be improved by dissection. It has the disadvantage that:

(*a*) A leakage post-operatively at this site is apt to be particularly serious, with a duodenal as well as a biliary fistula.

(*b*) *The anastomosis is rather close to the primary disease when the pancreas is carcinomatous.*

(*c*) The possibilities of alimentary reflux are real and a barium meal can often be seen entering the biliary channels.

For these reasons modern tendency is towards an increasing use of the jejunum, either as a loop defunctioned by enteroanastomosis or in the form of the Roux loop en-Y. The limb is usually brought up in front of the transverse colon and an easy anastomosis effected between the mobile gall bladder and the mobile intestine of similar diameter. When the common bile duct is used the union is made over a prosthesis such as a long arm T-tube or an indwelling temporary catheter which will pass on of its own accord or be withdrawn according to the Allen technique. In this case the jejunal end is closed around the duct and the union inverted as in the Kader-Senn "ink bottle" gastrostomy, or the jejunal end may be completely closed and the duct inserted into its side at the anti-mesenteric border The proximal cut end of jejunum is then anastomosed to the side of the jejunum some nine inches below the point of severance. It is important to close the gap between the upper lateral edge of jejunal mesentery and the mesentery of the distal jejunum

This operation takes longer and is more extensive than the more simple ones. Moreover, it involves radical interference with the alimentary tract and the chances of post-operative complications would seem to be greater. Such is not the case, however, and the results seem to justify its use when the expectation of life is greater than several

not depart from accepted principles in the management of simple ulcers. Diagnosis is unlikely to be complete unless surgery has become necessary and the ulcer has become available for histological examination. Symptoms may be caused by the actual bulk of heterotopic tissue acting as any other innocent tumour, occasionally causing an intussusception, and the treatment is similar. Rarely hyperinsulinism has been described, associated with an islet cell tumour in aberrant pancreatic tissue.

Injuries of the Pancreas

The surgeon is not often called upon to deal with a penetrating wound of the pancreas, for although in wartime surgery he should examine this organ in dealing with any upper abdominal wound, so many great vessels surround the gland that penetration is only too likely to be complicated by fatal hæmorrhage before surgery can be undertaken. A non-penetrating injury may occur in an accident on the road or playing field and may well be overlooked initially until contusion leads perhaps to hæmorrhage or necrosis, or later to the development of a false cyst. A third type of injury is that resulting from an operation on the stomach, duodenum, spleen, or bile passages. Cattell (1953) considers that these comprise the majority of injuries and lists the complications of pancreatic injury as follows:

(1) Acute pancreatic œdema and pancreatic necrosis.
(2) Immediate or delayed massive hæmorrhage.
(3) Pancreatic cyst.
(4) Pancreatic abscess
(5) Internal and external fistula.
(6) Pancreatic calculi
(7) Malnutrition.
(8) Diabetes mellitus.

Diagnosis is frequently difficult in the early stages, for the penetrating and non-penetrating injury is often complicated by injury to other structures whose manifestations dominate the clinical picture, while pancreatic injury at a major upper abdominal operation notoriously leads merely to a failure to make normal post-operative progress, accompanied by a singular paucity of abnormal symptoms and signs. If a pancreatic injury is suspected, estimation of the serum amylase may well provide crucial evidence allowing a positive diagnosis to be made

Treatment. The immediate treatment of a severe pancreatic injury is limited to securing hæmostasis and providing drainage. Later a traumatic cyst, or a fistula, may call for further surgery. The possibility of injury at operation should be remembered, particularly when dealing with the penetrating posterior ulcer of the stomach or duodenum and at splenectomy. If the gland has been accidentally injured, treatment of acute or chronic pancreatitis, or a traumatic cyst, follows the lines suggested later in this chapter. A pancreatic fistula should be given a chance to heal spontaneously, but eventually may call for implantation of the fistulous tract into the stomach or bowel or anastomosis of the pancreatic duct itself, mucosa to mucosa, to the jejunum, or distal pancreatic resection, before healing will occur

CHAPTER IV

SURGERY OF THE PANCREAS AND SPLEEN

RODNEY SMITH

THE PANCREAS

A mere 20 years or so ago the surgery of the pancreas contributed an almost insignificant proportion of the general surgeon's problems and text books of the early 1930's devote little space to this organ. Amid the dramatic progress shared in by the whole of surgery, the tremendous expansion in this particular field must be considered truly remarkable. It is now much less easy to cover all pancreatic lesions in a chapter of manageable proportions. The subject will be considered under the following headings:

(1) Congenital malformation of the pancreas.
(2) Injuries.
(3) Inflammations: (*a*) Acute pancreatitis.
(*b*) Chronic pancreatitis.
(4) Cysts.
(5) Carcinoma of the head of the pancreas and ampullary region.
(6) Carcinoma of the body and tail of the pancreas.
(7) Sarcoma and other rare tumours.
(8) Hyperinsulinism and Islet Cell Tumours.

CONGENITAL MALFORMATIONS

Various congenital malformations have been described and many of them eventually give rise to secondary pathological changes calling for surgical correction.

Annular Pancreas

An abnormal ring of pancreatic tissue, the duct system of which is very variable, partially or completely encircles the second part of the duodenum, the lumen of which is frequently narrowed and may even be the site of complete atresia. Symptoms, when present, are likely to be obstructive in character, though peptic ulceration of the stomach or duodenum is a not uncommon complication. Treatment is essentially surgical. Division of the aberrant pancreas risks laying open a large duct with a resultant pancreatic fistula, while it will not cure obstructive symptoms if the duodenum is itself the site of intrinsic stenosis. Short circuit by duodeno-jejunostomy is preferable to gastro-jejunostomy, but if a peptic ulcer is present partial gastrectomy is necessary.

Heterotopic Pancreas

Heterotopic pancreatic tissue may be found in the wall of the stomach or duodenum, less commonly in the jejunum, ileum, or a Meckel's diverticulum, very rarely in the colon, liver, bile passages, and other organs. Ulceration may lead to pain or hæmorrhage and when, as is usually the case, the stomach or duodenum is the site, treatment does

Investigations

Plain X-rays may show dilated coils of small bowel if paralytic ileus is present, or later the outline of a soft tissue mass. In an acute case it is not desirable to submit a very ill patient to a full examination with a barium meal or enema, but if the patient is seen for the first time some 10–14 days after the acute episode, with a palpable mass, barium studies may enable the relation to stomach, duodenum, and colon to be established. Examination of the blood may show leucocytosis or hæmo-concentration, depending upon the degree of infection or circulatory shock, but these findings are of course non-specific. Estimations of serum and urinary amylase are of fundamental importance and both should be done, for the elevation of the urinary amylase lags behind the serum amylase. After two to three days a fall to normal levels is usual even though clinical improvement may not follow. Elevation of the serum lipase and hyperglycæmia sometimes occur, but inconstantly.

Treatment. Until 20 years or so ago it was assumed that surgical intervention was necessary in acute pancreatitis. Since then it has become increasingly clear that if the surgeon does open the abdomen, there is little he can do apart from confirming the diagnosis and that the more energetic the local assault upon the pancreas, the higher the mortality is likely to be. Today it must be accepted that unless the diagnosis is in doubt, obstructive jaundice is present, or an abscess or cyst has developed, expectant treatment holds out the best chance of survival, and this consists of:

(*a*) The relief of pain. Morphine is usually ineffective and sometimes increases pain. Pethidine, if necessary in big doses, is preferable, but this too may prove ineffective and occasionally has even made the pain worse. No drug can be depended upon to control pain. Paravertebral injection of procaine has been used successfully and is well worth trying.

(*b*) The treatment of circulatory shock with intravenous blood, plasma, or electrolytic solutions.

(*c*) The anticipation and control of infection with antibiotics.

(*d*) The reduction of pancreatic secretion (i) by drugs; atropine, ephedrine, and banthine have all been used, or better probanthine 50 mgm. twice a day, (ii) continuous gastric suction should be used to keep the gastric secretion from the duodenal mucosa; ((iii) Irradiation of the pancreas has been suggested).

(*e*) The control with insulin in the rare complication of diabetes.

If the acute phase is survived, surgery may become necessary to deal with a cyst or abscess, or co-existent biliary disease.

If the abdomen has been opened on account of doubt in the diagnosis, minimal disturbance of the pancreas is advisable, measures being limited to the removal of completely detached fragments of sloughing pancreas and providing drainage of an abscess. The biliary system should not be interfered with unless obstructive jaundice or stones in the common bile duct call for choledochotomy.

CHRONIC PANCREATITIS

Two types of chronic pancreatitis may be recognized:

(*a*) *Chronic Fibroid Pancreatitis* may be produced by any lesion obstructing the main pancreatic duct and is thus common in cases of carcinoma of the head of the pancreas

INFLAMMATION

Acute Pancreatitis

Ætiology. The causes of acute pancreatitis still remain a matter for debate. Reflux of infected bile into the pancreatic duct system in the presence of a common channel of exit is generally accepted as a possible cause in some cases, precipitated perhaps by obstruction at the Ampulla of Vater by spasm or œdema of the sphincter of Oddi, or by a calculus. Other suggested causes include trauma, infection from the bowel lumen, lymph-borne or blood-borne infection, and local vascular accident, though this latter is more likely to be the result than the cause of acute pancreatitis.

Pathology. Although secondary bacterial invasion is common, the condition does not start as a true infection but as a disruption of pancreatic acini with the release into the parenchyma of the gland of digestive ferments, with consequent autolysis and digestion of blood vessels and local hæmorrhage. Characteristically, therefore, the gross features of acute pancreatitis are: œdema, hæmorrhage, fat necrosis, sloughing; later cellulitis and abscess formation.

Œdema, hæmorrhage, and sloughing should not be regarded as different varieties or stages of the disease, for all three are commonly seen at one and the same time in different areas of the gland. Fat necrosis is caused by the release of lipase which, travelling by blood or lymph vessels, produces the characteristic opaque white areas on the surface of the pancreas, peritoneum, mesentery, great omentum, and in other more distant sites.

Clinical Features

Symptoms vary enormously according to the severity and rate of the disruption of the gland. Typically, the patient is a fat, middle-aged person who after a heavy meal or over-indulgence in alcohol is suddenly seized with a very severe epigastric pain accompanied by a variable degree of shock and often cyanosis. The circulatory collapse may be so severe that coronary thrombosis is suspected, or if less marked the severity of the pain may lead to a diagnosis of perforated peptic ulcer or biliary colic. A less dramatic presentation with pyrexia suggests acute cholecystitis, particularly if a history of previous gall bladder disease is obtained. Later, as retroperitoneal digestion and hæmorrhage leads to paralytic ileus, a diagnosis of small bowel obstruction may be made. Later still, often not for a week or two, a tender epigastric mass may be present, as an abscess or cyst develops.

It is probable that apart from cases of severe pancreatic disruption presenting acutely in this way, some patients with undiagnosed episodes of less dramatic upper abdominal pain have in fact recovered spontaneously from acute pancreatitis. Recently more cases of this kind are being recognized.

Physical Signs also vary greatly. Tenderness, abdominal rigidity and guarding, circulatory shock and fever are common. Paralytic ileus is not usually manifest for 12–24 hours. Jaundice occurs in about one-fourth of all cases. Discoloration around the umbilicus (Cullen's sign) or in the flanks (Grey Turner's sign) is the result of extraperitoneal spread of extravasated blood. A palpable mass may be felt after about a week.



FIGS 114–115 *Chronic Relapsing Pancreatitis and Pancreatolithiasis* Specimen and X-ray of specimen removed by pancreato-duodenectomy, with relief from further attacks and return to normal health.

and ampullary region. In the past chronic fibroid pancreatitis has been considered by *some as itself a common cause of obstructive jaundice.* Most surgeons of experience in this field now believe that if laparotomy reveals biliary obstruction with a greatly



FIG. 113 X-ray (lateral) of patient suffering from severe relapsing pancreatitis and pancreatic calcification.

dilated thin-walled common bile duct and gall bladder, associated with chronic fibroid pancreatitis, the pancreatitis is most unlikely to be the cause of the jaundice, but the result of blockage of the pancreatic duct by a neoplasm, itself the cause of the jaundice.

(*b*) *Chronic Relapsing Pancreatitis* may give rise to recurrent attacks of typical acute pancreatitis or of less spectacular attacks of epigastric pain with variable gastro-intestinal symptoms and constitutional upset The condition is uncommon but probably accounts for more unexplained attacks of abdominal illness than has hitherto been recognized.

Treatment. Non-operative treatment consists of the control of the acute episodes as described above. There is little evidence that dieting and drugs have a significant effect upon the frequency and severity of attacks and surgical intervention of some kind is often necessary in the end. Various operations have been performed at one time or another for this condition, classified by Cattell (1953) as follows.





FIGS 114–115. *Chronic Relapsing Pancreatitis and Pancreatolithiasis* Specimen and X-ray of specimen removed by pancreato-duodenectomy, with relief from further attacks and return to normal health.

Indirect:

- A. Biliary tract procedures:
  1. Cholecystectomy—choledochostomy.
  2. Biliary-intestinal anastomosis.
  3. Sphincterotomy.
- B. Gastro-intestinal diversion:
  1. Gastroenterostomy.
  2. Pyloric exclusion.
  3. Gastrectomy.
- C. Nerve interruption:
  1. Sympathectomy.
     Thoracolumbar.
     Splanchnicectomy.
  2. Vagotomy.

Direct:

- A. Drainage of cysts.
- B. Pancreato-lithotomy.
- C. Anastomosis.
  1. Continuity.
  2. Diversion.
- D. Resection:
  1. Distal pancreatectomy.
  2. Pancreato-duodenectomy.
  3. Total pancreatectomy.

Cattell considers that cases without obstruction of the main pancreatic duct or with gross peripancreatic œdema are best treated by sphincterotomy, cases with the pathological process localized to the distal pancreas by distal pancreatectomy, and cases with obstruction of the main pancreatic duct at the head end by pancreato-duodenectomy if the surgeon and his team are experienced in this operation (Figs. 113–115).

Sphincterotomy. Exploration of the common bile duct is followed by estimation of the calibre of the sphincter of Oddi by passing graded dilators. The Doubillet sphincterotome (Fig. 116 (*a*) and (*b*)) is then passed, the sphincter cut and further dilators up to 8–10 mm. passed Alternatively, the sphincter may be divided under direct vision after duodenectomy.

PANCREATIC CYSTS

Various classifications have been adopted from time to time in describing pancreatic cysts.

The traditional division is into true and false cysts and if this is adopted, subdivision can be continued as follows·

True Cysts

1. Pancreatic cysts accompanying polycystic disease of other organs.
2. Fibrocystic disease
3. Retention cysts.
4. Dermoid cysts.

5. Hydatid cysts.
6. Angiomatous cysts:
   (*a*) Hæmangioma.
   (*b*) Lymphangioma.
7. Neoplasm:
   (*a*) Cystadenoma and cystadeno-carcinoma.
   (*b*) Teratoma.
   (c) Other neoplasms with cystic degeneration.

False Cysts

1. Of inflammatory origin.
2. Of traumatic origin.

Multiple small cysts need not be further discussed. True and false single cysts are best considered together as regards diagnosis for the clinical manifestations have many similarities as well as some differences. The true cyst frequently grows slowly over the course of several years before causing symptoms, while a false cyst is likely to become apparent soon after an attack of pancreatitis or trauma to the gland. The true cyst

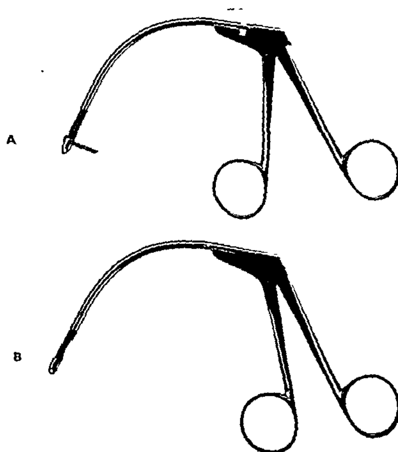


FIG 116. The Doubilet sphincterotome with the blade open (*a*), and closed (*b*).

usually projects forwards into the lesser sac and then presents above the stomach through the lesser omentum, or less commonly between the stomach and colon through the gastro-colic ligament. The false cyst is usually a cyst of the lesser sac but may burrow behind the posterior peritoneum, extend into the loin, between the layers of the greater omentum, or even up into the mediastinum.

The patient may come to his doctor complaining of an abdominal mass, or of epigastric pain, which is a very common symptom even though it is not often severe.



(From the "Surgery of Pancreatic Neoplasms" by Rodney Smith E. & S. Livingstone Ltd., Edinburgh)

FIG 117 *Cystadeno-carcinoma of the pancreas* X-ray showing a spherical soft tissue shadow in the region of the pancreas

Nausea, vomiting, and alteration of bowel habit sometimes occur. Obstruction of the common bile duct with jaundice may result and if the cyst grows to a sufficient size obstruction of the duodenum, jejunum, and even colon.

Physical signs are related to the abdominal mass which is usually smooth and spherical, though sometimes lobulated or irregular. Tenderness is a sign of inflammation or necrosis, while the cystic nature of the swelling is detectable in rather more than 50 per cent of cases. Most cysts are fixed but true cysts of the tail of the gland are sometimes mobile.

Investigations

Plain X-rays may show a soft tissue shadow and sometimes calcification in the wall of the cyst (Fig. 117). Barium studies may show displacement or a smooth indentation of the stomach, small bowel or colon. Intravenous pyelography may demonstrate displacement of the left kidney. Estimations of serum and urinary diastase only occasionally show



(From the "Surgery of Pancreatic Neoplasms" by Rodney Smith, E & S Livingstone Ltd, Edinburgh)

FIG 118 *Cystadeno-carcinoma of the pancreas* The tumour dissected out of the mass removed at operation (resection of spleen and of pancreas distal to the head) Patient in excellent health five years later.

any abnormality. Estimation of blood sugar occasionally reveals diabetes mellitus, which Cattell considers accompanies the pancreatic cyst in 10–15 per cent of cases.

Treatment. True cysts should be removed by whatever surgical method appears appropriate. Enucleation is rarely advisable unless malignancy can be rigidly excluded, and segmental pancreatic resection with suture of the gland should never be attempted for it is inevitably followed by pathological changes in the distal segment and usually by a pancreatic fistula. Cysts in the head may necessitate pancreato-duodenectomy, but if in the body or tail can be excised by the less extensive operation of distal pancreatectomy, (Figs 117–118).

Several methods of surgical treatment are available for dealing with false cysts. *Excision* is rarely sound in theory or simple in practise, for there is no true cyst lining and the wall, consisting usually of the boundaries of the lesser sac, is intimately attached to a number of large vessels. Early attempts at excision not infrequently led to dangerous

hæmorrhage and spectacular discomfiture of the surgeon, and thus to a search for less hazardous methods of cure.

Marsupialization was historically the next stage in the development of technique, the cyst being opened, evacuated, packed with gauze, and the edges sewn to the edges of the skin wound. This procedure is still practised and cures many cysts, but in a fair number of cases a fistula persists, or if this closes the cyst recurs.

Internal drainage developed from the necessity in some cases of implanting into the gastro-intestinal tract the fistula sometimes left after marsupialization has failed to cure the cyst. As Milroy Paul (1949) has pointed out, a false cyst is usually a cyst of the lesser sac and so densely adherent to the back of the stomach that anastomosis of the cyst to the stomach is a simple operation, if performed through the lumen of the latter. The anterior wall of the stomach is incised and a disc cut out of the posterior wall to enter the cyst. A few sutures through all coats secure hæmostasis, but none need be inserted for the purpose of anastomosis since the cyst and stomach are so densely adherent. The anterior wall of the stomach and the abdominal wound are now closed. Other varieties of internal drainage occasionally practised are cysto-duodenostomy and cysto-jejunostomy.

Pancreato-gastrostomy. A false cyst, after an injury to the main pancreatic duct is sometimes best treated by exploration of the cyst and identification of the opening in the duct, which is anastomosed to the stomach, or less frequently to the duodenum or jejunum.

Distal Pancreatectomy. A false cyst taking origin from a grossly disorganized distal pancreas is best treated where possible by excision of the pathological segment of the gland.

Present day views on the treatment of false cysts may be summarized as follows: marsupialization is not often the best treatment available; internal drainage is an attractive alternative and quite often the most appropriate procedure. Nevertheless, this operation leaves undisturbed the cause of the cyst and when possible exploration is preferable in the hope that a more definitive operation, such as distal pancreatectomy, or pancreato-gastrostomy may be carried out.

CARCINOMA OF THE HEAD OF THE PANCREAS AND AMPULLA OF VATER

Whilst certain differences exist in diagnosis, treatment and certainly prognosis, between carcinomas in these two sites, there are many similarities and they are best considered together.

Pathology. The term ampullary carcinoma is usually held to embrace all carcinomas of a true ampulla of Vater and the immediate peri-ampullary region, thus carcinomas arising from the terminal common bile duct, terminal pancreatic duct and from the duodenal mucous membrane over the papilla of Vater. *Carcinoma of the head of the pancreas* is usually ductal rather than acinar in origin. It causes a hard, irregular enlargement of this part of the gland, the growth soon involving the terminal common bile duct and pancreatic duct Carcinoma of the ampulla or head of the pancreas thus causes early obstruction of the terminal common bile duct with dilatation of the biliary tree proximally, including the gall bladder Obstruction of the main pancreatic duct similarly leads to dilatation of the duct system and ofte

Spread of the Tumour

Direct spread of an ampullary tumour soon leads to ulceration into the duodenum and eventually the growth may encircle the lumen and cause obstruction. Spread into the duodenum is less common and later in carcinomas of the head of the pancreas and the tumour extends outside the confines of the gland itself so that the superior mesenteric vessels or portal vein, stomach or inferior vena cava may become involved.

Lymphatic spread takes place to nodes between the head and the second part of the duodenum and to the subpyloric group. Later cœliac and para-aortic nodes and nodes in the portal fissure are involved. Occasionally mediastinal and supraclavicular nodes may contain metastases. Blood-borne metastases appear first in the liver and later and less often in lungs, brain, skin, and skeleton. Occasionally a tumour composed of functioning pancreatic cells releases lipase into the blood stream and areas of subcutaneous fat necrosis and painful effusions into the joints produces a puzzling clinical picture.

Signs and Symptoms. Diagnosis. The clinical features of carcinoma of the ampulla and of the head of the pancreas are similar but there are certain important differences.

AMPULLARY CARCINOMA

An ampullary carcinoma nearly always obstructs the common bile duct while it is still very small and, therefore, the early onset of obstructive jaundice without other important symptoms is common. With the jaundice occur nausea, anorexia, and therefore loss of weight, a dirty tongue, and offensive breath, bradycardia, pruritus, bile in the urine and acholic stools. There may be melæna, and sometimes the altered blood gives to the acholic stool a metallic or silvery appearance. Pain is a symptom in about 50 per cent of cases, is seldom severe and not often precedes jaundice in onset. Usually the jaundice deepens progressively without remissions but some variation should not be held to exclude an ampullary tumour. One patient of the writer was jaundiced for two weeks with a palpable gall bladder but, admitted to hospital one week later, had almost lost his jaundice, which cleared completely while he was being investigated, laparotomy nevertheless disclosing an ampullary carcinoma which was successfully resected. Very occasionally, an ampullary tumour presents with obstruction of the second part of the duodenum or an unexplained anæmia. Steatorrhœa is infrequent, caused by obstruction of the pancreatic duct. Abnormal physical signs relate in the main to the dilatation of the biliary tract above the obstruction. The liver is enlarged and smooth with a rounded edge; it is only slightly tender unless obstruction has led to ascending cholangitis. A *palpable distended gall bladder* is an important finding, for as Courvoisier stated, obstruction of the common bile duct by a gall stone very seldom produces a palpable gall bladder. Thus, a palpable gall bladder in the presence of obstructive jaundice nearly always means malignancy at the lower end of the common bile duct (Fig. 117). The reverse is not true; absence of a palpable gall bladder does not exclude malignancy, for previous chronic cholecystitis may have left an inelastic gall bladder, or the tumour may have extended up the common bile duct to involve the junction with the cystic duct. Later physical signs relate to malignant deposits in lymph nodes, liver or peritoneal cavity.

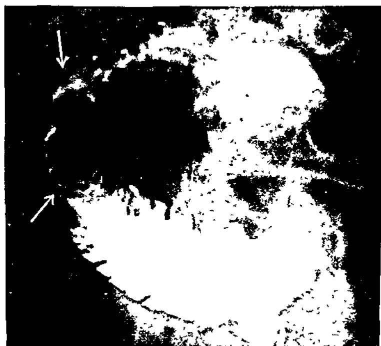
Investigations

Various liver function tests are of some value in confirming the obstructive character of the jaundice. Thus a marked elevation of the serum alkaline phosphatase and a



(From the "Surgery of Pancreatic Neoplasms" by Rodney Smith, E. & S. Livingstone Ltd., Edinburgh)

FIG. 119 The dilated gall bladder caused by malignant obstruction of the common bile duct



(From the "Surgery of Pancreatic Neoplasms" by Rodney Smith, E. & S. Livingstone Ltd., Edinburgh)

FIG 120 Barium meal showing the "Reversed 3" deformity of the duodenal mucosa caused by an ampullary new growth

normal thymol turbidity indicate obstructive jaundice, while a moderate elevation of the serum alkaline phosphatase and a high thymol turbidity suggest a hepatogenous jaundice. Tests for occult blood in the stools are positive in most cases of ampullary carcinoma.

X-rays

A barium meal may possibly show a distorted or ulcerated duodenum at the level of the ampulla. Sometimes a combination of œdema and infiltration gives rise to the characteristic "reversed 3" deformity of the duodenal mucosa (Fig. 118). Transhepatic cholangiography (Nurick 1953, Rodney Smith 1954) (Figs. 119–122), will show a biliary system obstructed at the lower end of the common bile duct, if this investigation is considered justifiable. The final investigation is laparotomy, and too much time should not be wasted in complex investigations once a diagnosis of obstructive jaundice has been made.

CARCINOMA OF THE HEAD OF THE PANCREAS

The diagnostic features are similar to those of the ampullary carcinoma with certain important differences. *Pain* is a very common symptom, may be severe and more often than not precedes jaundice in onset. Typically, it is epigastric in site, referred through to the back and affected by posture, being worse when lying down at night. Quite often a less clear cut description is obtained of an ill-localized pain, referred sometimes to the sternal or submammary region or of a girdle distribution. The rapidity with which *jaundice* develops depends upon how far the tumour in the pancreatic substance must extend before reaching the common bile duct. The jaundice is likely to be accompanied by a *palpable gall bladder*. *Occult blood* in the stools is a less frequent finding than in ampullary carcinoma. The *barium meal* may show widening of the duodenal loop but as this loop is in any event of variable width and may be widened by other lesions than an enlarged pancreatic head (e.g. enlarged lymph nodes, the site of secondary carcinoma or Hodgkin's disease) evidence of duodenal invasion is necessary before malignancy can be reported with conviction.

Treatment

Preparation should include correction of anæmia, dehydration and electrolytic imbalance and the administration of Vitamin K, either by injection or orally with bile salts.

The abdomen opened, a dilated gall bladder usually presents even if, lying under an enlarged liver, it had not been palpable pre-operatively. Exposure of the common bile duct finds this similarly dilated and nearly always thin-walled and without œdema or inflammation. Examination of the region of the lower end of the common bile duct and head of pancreas is now carried out, the peritoneum lateral to the second part of the duodenum being incised so that the head of the pancreas can be lifted forwards and examined between finger and thumb. The findings conform to one of four general patterns:

1. An obvious pancreatic carcinoma is present giving rise to the typical hard, craggy, irregular enlargement.
2. An ampullary new growth can be felt through the duodenal wall.
3. No abnormality of the pancreas or ampulla is palpable.
4. The head and a variable part of the body and part of the pancreas are firm and indurated but no obvious carcinoma can be detected.

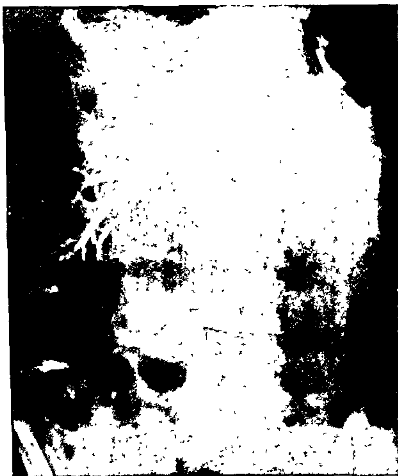


FIG 121. Percutaneous transhepatic cholangiogram showing gross dilatation and obstruction of the common bile duct (Ampullary new growth)



FIG 122. For comparison with Fig 121, percutaneous transhepatic [illegible]

flow, and incomplete obstruction of the right main hepatic duct (Secondary carcinoma from a primary in the breast excised five years previously.)



FIG 123 Percutaneous transhepatic cholangiogram showing gross dilatation and obstruction of the common bile duct.



FIG. 124 Barium meal following up the cholangiogram of Fig. · The common bile duct is obstructed above the level of the duode cap, therefore *not* by an ampullary new growth but by a grov well up the common bile duct or in the pancreas

The problem which has troubled surgeons for years is how to proceed if obstructive jaundice is clear but no carcinoma demonstrable. It now seems clear that (*a*) almost invariably if the gall bladder and common bile duct are not only dilated but thin-walled and without evidence of past or present inflammation, an early new growth is present and biliary short-circuit is likely to be followed by later recurrence of symptoms and development of metastases. The interval may be a matter of months or as much as two to three years. (*b*) Chronic pancreatitis and obstructive jaundice may occasionally be present without a carcinoma, but nearly always evidence of marked biliary infection is found in such cases, the bile passages being thick-walled and the gastro-hepatic omentum œdematous.

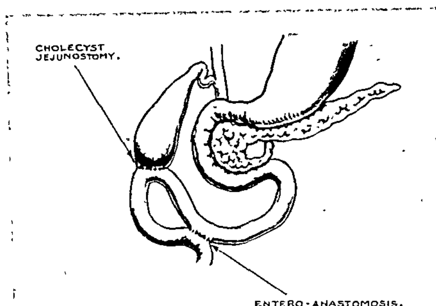


(From the "Surgery of Pancreatic Neoplasms" by Rodney Smith, E. & S. Livingstone Ltd., Edinburgh)

Fig. 125 Tiny primary carcinoma of the ampullary type lying between the pancreas and the duodenal mucosa, causing only transient jaundice. Resection of this type of tumour may well be followed by prolonged survival, but is unlikely to be achieved unless the surgeon is willing to open the abdomen and explore carefully the ampullary region on quite slender evidence.

In the past pancreatic biopsy has seemed a justifiable step, but unfortunately surface biopsy may easily miss a carcinoma if deeply placed and carries a high risk of causing a pancreatic fistula. Transduodenal biopsy (Rodney Smith 1953) avoids these two disadvantages but carries a possible risk of dissemination of the tumour. The writer identified by transduodenal biopsy a tiny new growth of the ampulla (Fig. 125) treated then by radical pancreato-duodenectomy. One year later a recurrence in the wound appeared, strongly suggesting local implantation at operation. Richard Cattell of Boston (1954), whose experience of pancreatic carcinomas is unrivalled, considers that the obstructed, thin-walled, dilated biliary system produced by a new growth at the

lower end of the common bile duct can be recognized at sight, and that in suitable cases resection should be carried out without trans-duodenal exposure of the ampulla even if no new growth can itself be palpated. Fibroid pancreatitis is quite commonly present and obstruction of the main pancreatic duct is frequently demonstrable by palpating



(From the "Surgery of Pancreatic Neoplasms" by Rodney Smith, E & S Livingstone Ltd, Edinburgh)

FIG 126 Internal biliary drainage to palliate the jaundice caused by an [illegible]

the pancreatic substance in which the dilated duct stands out, feeling like the inner tube of a fountain pen.

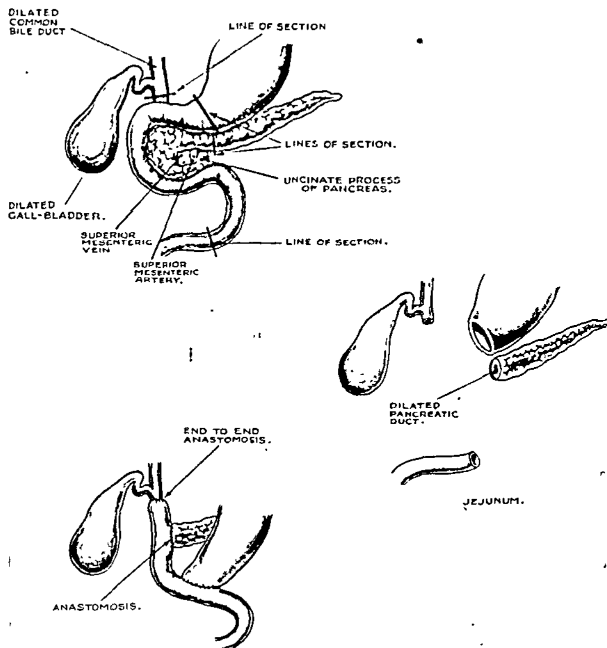
After preliminary exploration the following lines of treatment are available:

1. Closure of the abdomen without further surgery.
2. Palliative Surgery.
3. Conservative Resection.
4. Radical Resection.

Widespread intraperitoneal metastases, malignant ascites or malignant extension into the portal fissure obstructing the hepatic ducts usually contra-indicate further surgery. Early hepatic metastases or local fixity call for palliative surgery to relieve obstructive jaundice for although the period of survival may not be significantly prolonged the patient is usually made very much more comfortable. Various palliative operations have been performed from time to time.

External biliary drainage. Cholecystostomy is seldom the best palliative operation. It relieves the jaundice but of course leaves the patients with a permanent total biliary fistula. It is occasionally the most suitable first stage in a two-stage resection but only if (*a*) it is tolerably certain that resection is technically possible, and (*b*) the patient is in such poor state that no more extensive procedure can be carried out.

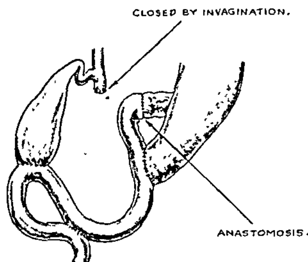
Internal biliary drainage is a different matter and can be achieved by anastomosing the gall bladder or common bile duct to the stomach or small intestine. One risk of this operation is that food or intestinal contents may reflux into the bile passages and cause ascending cholangitis. For this and for certain technical reasons the most suitable biliary short circuit is cholecyst-jejunostomy, either performed En-Y or as a side-to-side anastomosis with an entero-anastomosis of afferent and efferent jejunal loops placed 8 in. from the biliary anastomosis (Fig. 126).



(From the "Surgery of Pancreatic Neoplasms" by Rodney Smith, E & S Livingstone Ltd., Edinburgh)

Fig 127 One-stage radical pancreato-duodenectomy Diagram showing extent of resection and reconstruction favoured by the writer Anastomosis over rubber tube

If no metastases are present, carcinoma of the ampullary region should be treated by radical pancreato-duodenectomy, the extent of this resection and the writer's usual reconstruction being shown in Figs. 127–128. Where possible this operation is best performed in one stage. Less frequently a two-stage procedure is indicated. The results of



(From the "Surgery of Pancreatic Neoplasms" by Rodney Smith, E. & S. Livingstone Ltd., Edinburgh)

FIG 128 Two-stage radical pancreato-duodenectomy Diagram showing procedure favoured by the writer Anastomosis over rubber tube

radical resection for ampullary carcinoma are not discouraging, for the operation does not carry a prohibitive mortality and the chances of long term survival are reasonably good (*see* Tables 1 and 2).

Occasionally a small ampullary tumour in an aged patient is justifiably treated by conservative, local resection. Gordon-Taylor had several successes with transduodenal resection, before advances in anæsthesia and anti-shock measures made the radical operation less hazardous.

TABLE 1. MORTALITY OF RADICAL PANCREATO-DUODENECTOMY FOR CANCER

	Cases	*Deaths*	*Percentage*
Cattell (1953)			
Carcinoma of Head of Pancreas	46	2	17·3
Ampullary Carcinoma	30	2	6 6
Rob and Rodney Smith (1954)			
Carcinoma of Head of Pancreas	9	3	33
Ampullary Carcinoma	21	1	4 7

TABLE 2. SURVIVAL RATES AFTER RADICAL PANCREATO-DUODENECTOMY CATTELL (1953)

For Carcinoma of Head of Pancreas	5 per cent of patients survived 5 years.
For Ampullary Carcinoma	[illegible]

Carcinoma of the head of the pancreas should be treated either by radical pancreato-duodenectomy or by biliary short circuit. Unfortunately the results of the radical operation are not nearly so good as in ampullary carcinoma. The immediate mortality is much higher and the chances of prolonged survival much smaller (Tables 1 and 2). For this reason, whilst exploration and mobilization of the duodenum and pancreas are started with the intention of carrying out a radical resection, if dissection in the peri-pancreatic planes is hampered by fixity or œdema, the operation should be abandoned and palliative short-circuit performed instead.

TOTAL PANCREATECTOMY

Total excision of the pancreas is not technically a much more extensive procedure than radical pancreato-duodenectomy. Some surgeons have argued that division of the pancreas may well cut across a pancreatic duct full of free malignant cells and that total pancreatectomy, by avoiding this, may well give improved long term results.

In practice total pancreatectomy carries a very high immediate mortality and although, if survival occurs, the degree of diabetes developing does not usually necessitate big doses of insulin, the metabolic problems are often far from uncomplicated. Moreover, there is little evidence that the long term results after total pancreatectomy are any better than after pancreato-duodenectomy.

TECHNIQUE OF RADICAL PANCREATO-DUODENECTOMY

Unless care is taken it is possible to get into quite serious trouble with this operation, and technique should be so designed that no irrevocable step is taken until the removability of the tumour has been conclusively demonstrated. It is, to say the least of it, embarrassing to divide the common bile duct, stomach, and pancreatic body and then find that, the portal vein being invaded by growth, a position has been reached from which it appears impossible either to advance or retreat.

Thus, the surgeon should first mobilize the duodenum by division of the peritoneum lateral to it. Lifting forward the whole duodenal curve with the head of the pancreas, he can show that the inferior vena cava is not implicated.

Anteriorily the lesser sac is opened dividing the gastro-colic omentum, and the front of the pancreas is cleared so that the superior mesenteric vessels are displayed crossing the uncinate process and the third part of the duodenum.

Attention is now switched to the structures in the free edge of the lesser omentum. The peritoneum is incised and the dilated common bile duct encircled with a tape; the gastro-duodenal and right gastric arteries are identified above the pylorus and divided between ligatures. The common bile duct is retracted to the right, and the front of the portal vein is exposed as it emerges behind the pancreatic head.

The left index finger now gently opens up the plane between the portal vein and the back of the pancreas; at the same time the right index finger is passed up in front of the superior mesenteric vein and behind the pancreas. No force must be used in the manœuvre. If the two fingers encounter no area of posterior malignant infiltration and can be made to meet with the pancreas in front and the portal venous system behind, pancreato-duodenectomy is technically possible and the operation can proceed.

The common bile duct is divided and, using a sucker, the dilated extra-hepatic biliary system is emptied, after which further leakage of bile into the operative field is prevented

by a bulldog clip. The antrum is divided between Kocher clamps, the proximal of which is covered by a strip of gauze and tucked away in the left abdomen. The pancreatic body is divided and hæmostasis secured by under-running any blood vessels with silk. The portal vein and its tributaries are exposed but not disturbed at this point. The transverse colon is now elevated and, the ligament of Treitz being incised, the duodeno-jejunal flexure is fully exposed. The jejunum is divided between clamps and the proximal end passed up through the hole in the mesocolon. Dissection is now directed once more above



(From the "Surgery of Pancreatic Neoplasms" by Rodney Smith, E. & S. Livingstone Ltd., Edinburgh)

FIG 129. Radical pancreato-duodenectomy. Extent of the resection as shown by an operative specimen.

the colon. The distal duodenum and divided proximal jejunum are extracted from beneath the superior mesenteric vessels, the upper jejunal branches and the inferior pancreato-duodenal artery being divided and tied off with fine silk.

Removal of the mass is now prevented only by the attachments between the head and uncinate process of the pancreas and the portal and superior mesenteric veins. These attachments conceal the pancreatic cervical vein entering the right side of the origin of the portal vein from the neck of the pancreas, the inferior pancreato-duodenal vein entering the superior mesenteric vein from the groove between the duodenum and pancreatic head, and several unnamed short stout veins running directly from the uncinate process into the superior mesenteric vein on its postero-lateral aspect. The first two of these must be identified and divided between silk ligatures. The smaller veins are difficult

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The common bile duct is divided and, using a sucker, the dilated extra-hepatic biliary system is emptied, after which further leakage of bile into the operative field is prevented

from the biliary-jejunal anastomosis in order to minimize the risk of ascending cholangitis.

The abdomen is now closed with drainage.

THE TECHNIQUE OF TOTAL PANCREATECTOMY (Fig. 130)

The technique is identical with that of radical pancreato-duodenectomy except that instead of dividing the pancreas the gland is mobilized distally as far as the spleen. This too is mobilized and removed with the pancreas after ligation and division of its vessels. Naturally only two and not three reconstructive anastomoses are now required.

Careful control of the inevitable post-operative diabetes is necessary. The difficulty usually lies in the fluctuating and unpredictable requirements of insulin rather than in the severity of the diabetes produced.

CARCINOMA OF THE BODY AND TAIL OF THE PANCREAS

Carcinoma of the body or tail of the pancreas is less common than that arising in the head. On purely anatomical grounds it might be imagined that resection would prove simpler, for distal pancreatectomy, leaving undisturbed the head of the gland in the duodenal loop, presents few technical difficulties and there is no complicated reconstruction of gastro-intestinal and biliary systems to follow. Unfortunately this anatomical advantage is more than offset by the difficulty in making an early diagnosis.

Diagnosis. The clinical story is one of pain, often severe, in the epigastrium, back or chest, worst at night or on lying down and little affected by food, accompanied by loss of weight and loss of appetite and other rather more indeterminate symptoms such as flatulence, dyspepsia, alteration of bowel habit, lassitude and asthenia. It is only too easy after a barium meal, barium enema and cholecystogram have all proved normal to label the patient "neurosis only." A palpable mass sometimes presents but usually means inoperability. Spontaneous venous thrombosis which may occasionally occur in any carcinoma occurs more frequently with pancreatic carcinoma than in other sites. Late invasion or obstruction of the duodenum may cause obstructive vomiting and the tumour may eventually reach the head end of the gland and obstruct the common bile duct.

A barium meal will show nothing in the early stages but later extrinsic pressure upon, or invasion of the stomach or duodenum may be demonstrable.

Treatment. Resection by distal pancreatectomy should be carried out where possible. If the tumour is locally fixed and irremovable, obstruction of the duodenum or common bile duct may occasionally call for a palliative short-circuiting operation.

The Technique of Distal Pancreatectomy

The lesser sac is opened by division of the gastro-colic ligament and, assuming that the stomach is not invaded by growth, the greater curvature is separated completely from its attachments to the spleen. This organ is itself mobilized by division of the postero-lateral peritoneal reflections and drawn into the wound with the tail of the pancreas. As the mobilization of the pancreas proceeds proximally, the splenic vessels are put on the stretch, ligated and divided.

to secure unless a thin sliver of pancreas is left along the vein, clamped and tied as the mass is dissected away from the vein. The extent of the mass removed is shown in Fig 129.

Reconstruction starts with end-to-end anastomosis of the common bile duct to part of the open end of the jejunum, the rest of the circumference of which is closed. This junction may, or may not, be made over a rubber tube, depending upon the preference of the surgeon and the fragility of the thinned and dilated common bile duct. The pancreatic stump is prepared for anastomosis by inserting interlocking mattress sutures of

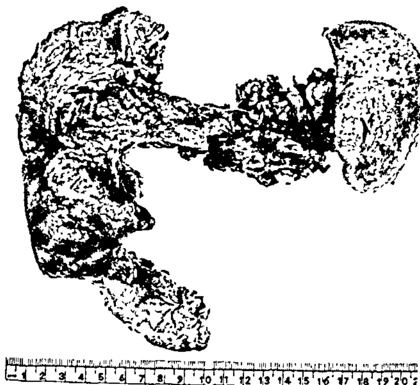
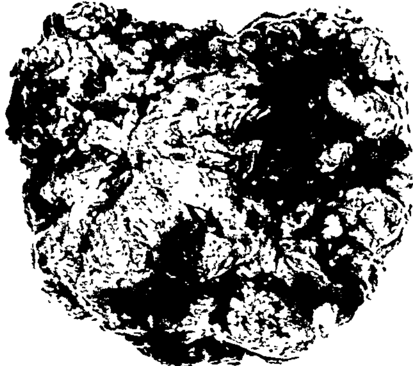


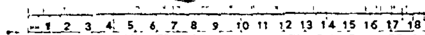
FIG 130 Total pancreatectomy Extent of the resection as shown by an operative specimen

silk to compress and secure absolute dryness and hæmostasis of the raw surface. Care is taken not to compress the duct of Wirsung, which is nearly always dilated and easily identifiable Incision through the sero-muscular layer of the jejunum allows exposure of an oval area of mucosa the shape and size of the cross-section of the pancreas.

Using a continuous silk suture the capsule of the pancreas is sewn to the cut sero-muscular margin of the jejunum Inside this the cut pancreatic surface becomes opposed to the jejunal mucosa In this a small puncture has been made opposite the duct of Wirsung and a fine rubber tube inserted so that one end projects 1 in. into the jejunal lumen and the other end one inch into the pancreatic duct. This tube is anchored by a single catgut stitch through the duct wall, tube, and the jejunal mucosa.

The third anastomosis to be constructed is between the stomach and the side of the jejunum The technique does not differ from that employed in any standard gastrectomy and need not be further discussed, except to add that it should be placed a good 8 in.







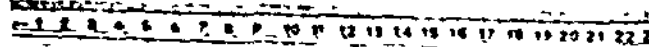


FIG 133–134. Spindle-celled sarcoma of the pancreas. Dissection of the mass removed showing the central soft fleshy growth and the surrounding implicated viscera



Fig. 131. Spindle-celled sarcoma of the pancreas Photograph at operation (taken from the head end of the table) showing the enormous tumour bulging through the gastrocolic omentum



Fig. 132. Spindle-celled sarcoma of the pancreas. *Photograph at operation* (taken from the foot of the table). Mobilization of spleen and division of pancreas, duodenum, and stomach just below the cardia leaves the tumour attached only to the transverse colon and meso-colon

to normal as the attack is cut short with sugar. The three essentials on which diagnosis rests, often referred to as Whipple's essential triad are:

1. Attacks of spontaneous hypoglycæmia induced by fasting or exercise.
2. A blood sugar during attacks below 50 mgm. per 100 ml.
3. Prompt relief of an attack by giving sugar.

In completing the investigations, the possibility of pathology in other ductless glands should not be forgotten, but in general a patient satisfying Whipple's essential triad should be explored for an islet cell tumour.

Surgical Treatment. At operation the whole gland may have to be mobilized before the tumour is found, and in any case even with one tumour found the surgeon must make sure that it is single. Removal is effected by enucleation or wedge resection. Post-operatively the blood sugar often soars, but even if it reaches 400–500 mgm. per 100 ml. no alarm need be felt and in the absence of ketosis no insulin need be given. The level returns to normal within a day or two and it may well be that in the presence of a tumour producing insulin in excess of the body's normal requirements, the islets become functionally dormant and do not immediately become active when the tumour is removed and that the stimulus necessary to restore activity is hyperglycæmia. The results of excision of the tumour are excellent.

If no tumour can be found, subtotal pancreatectomy is usually performed, removing the whole of the gland distal to the head, for it is claimed that a diffuse hyperplasia of the islets of Langerhans can produce the same effects as a tumour. If the excised specimen is found on examination to contain a tumour deep in its substance, the result of the operation is likely to be excellent, but if not the result is much more problematical and the chances of a cure are about the same whether histological examination of the specimen shows hyperplastic or normal islets.

References

Cattell, R. B and Warren, K. W. (1953) *Surgery of the Pancreas*, Saunders, Philadelphia.
Cattell, R. B. (1954) Personal Communication.
Nurick, A W, Patey, D. H and Whiteside, G C. G (1953) *Brit. J. Surg.* 41, 27.
Paul, M. (1949) *Brit J Surg.* 37, 99
Rodney Smith (1953) *The Surgery of Pancreatic Neoplasms*, E. and S. Livingstone, Edinburgh.

THE SPLEEN

For all practical purposes the only operation performed upon the spleen is splenectomy. The indications may be roughly grouped together under two headings, medical and surgical, and sub-divided thus

MEDICAL INDICATIONS

1. Familial Hæmolytic Anæmia.
2. Acquired Hæmolytic Icterus.
3. Idiopathic Thrombocytopenic Purpura
4. Hypersplenism.

Apart from malignant infiltration there is no difficulty in lifting forward the whole of the pancreas as far proximally as the neck. Division is made well clear of the lesion and the cut surface is closed with a series of interlocking mattress sutures of silk.

Rare Tumours

The first pancreatic tumour resected is said to have been a *spindle-celled sarcoma* removed by Trendelenburg in 1882. This is even rarer than *lympho-sarcoma.* One example of each of these has been encountered by the writer (Figs. 129–132). Other rare tumours which have been described include adenoma, melanoma, neurofibroma, lymphangioma, carcinoid, lipoma, fibroma, myxoma, chondroma, perithelioma. In most cases the discovery of a neoplasm calls for resection if technically possible, for the chances of it being innocent are slender.

Islet Cell Tumours and Hyperinsulinism

The existence of these little tumours has been recognized since the beginning of the century, but naturally enough their physiological activity was not appreciated until the 1920's, when insulin was isolated and its properties demonstrated.

Pathology. The islet cell tumour is usually small, often not more than 5 mm. in diameter, encapsulated and single, though two or more occasionally co-exist. It may occur anywhere in the pancreas but most commonly projects slightly from the anterior surface of the body, being rather darker in colour and firmer in texture than the rest of the gland, and covered by a network of little vessels. Microscopically it is composed of clumps or strands of cells, usually resembling islet beta cells, and although absence of a proper capsule in places and an appearance very like infiltration of the surrounding pancreas may suggest malignancy, practically all tumours are adenomas and benign, though an occasional malignant islet cell tumour, even with metastasis, has been described.

Diagnosis. The story told by a patient with a functioning islet cell tumour is very characteristic. Attacks of spontaneous hypoglycæmia occur when fasting or after exercise, thus commonly in the early morning or when rising before breakfast. Dizziness, faintness, and sweating with inco-ordination of speech or gait are followed by drowsiness deepening to unconsciousness with convulsions, frothing at the mouth and incontinence. Sometimes only mild symptoms occur but an attack may be severe enough to threaten life. A diagnosis of epilepsy or psychosis is often made, but frequently the patient provides the key to diagnosis by volunteering that food or a sweet drink may prevent or abort an attack. Physical examination is usually negative, though many patients have gained weight on account of the effect of excess insulin upon appetite and the patient's own experience that food may prevent attacks. An attempt to clinch the diagnosis should be made with a simple test A sample of blood for estimation of sugar is taken one morning from the fasting patient who is then gently exercised until symptoms occur; a second sample of blood is taken and the attack cut short by giving sugar by mouth or vein, after which a final sample of blood is taken. The classic findings are: a low fasting blood sugar, often somewhere between 50 and 90 mgm per 100 ml., a very low blood sugar at the onset of symptoms, 30 mgm or even lower having been described, and return to the previous level as the attack is stopped by giving sugar. Electro-encephalographic tracings are of interest and some value during this test, abnormal tracings of no particular diagnostic pattern being obtained during the hypoglycæmic attacks, returning

red blood cells coated with the globulin antibody precipitate out in clumps when treated with this serum.

Treatment. Two factors in treatment may prove effective; splenectomy and the administration of cortisone or ACTH. *Cortisone and ACTH* will bring about a dramatic remission in almost every case, and there is no reason why continued administration should not hold the condition in check indefinitely. It is, however, undesirable for the patient to live on cortisone for ever and hormone therapy should be employed in order to produce a remission during which splenectomy can be performed. *Splenectomy* is not the certain cure that it is in familial hæmolytic anæmia. It is nevertheless well worth doing in cases of idiopathic acquired hæmolytic icterus, about half of which are cured by operation or materially benefited. Some cases, however, are secondary to other causes of splenomegaly, such as Hodgkin's disease, leukæmia or reticulosarcoma, and in these splenectomy is less likely to be helpful and is rarely indicated. Blood transfusion may supplement hormone therapy or surgery if a dangerous level of anæmia exists, but it must be remembered that in severe episodes rapid sensitization of donor cells may occur and the transfused blood may be largely destroyed within a few days. (Loutit and Mollison, 1946).

Idiopathic Thrombocytopenic Purpura

Thrombocytopenic purpura may similarly be idiopathic or secondary to leukæmia, aplastic anæmia or the action of various drugs. Examination of blood and the bone marrow by sternal puncture must establish an accurate diagnosis, for splenectomy is only of benefit in the idiopathic variety, where some 60 per cent are cured and 20 per cent greatly improved (Hynes 1954). The other 20 per cent are not improved and unfortunately there is no way of picking out these unfavourable cases before operation.

The cause of the condition is not known although it has been suggested that an antibody affecting platelets is present analogous to that affecting red cells in hæmolytic icterus. As in this latter disease, Cortisone and ACTH both are likely to bring about a remission.

Treatment. Idiopathic thrombocytopenic purpura is seen most commonly in children, in whom prolonged spontaneous remissions frequently occur and occasionally complete spontaneous cure. For that reason there should be no haste in advising surgery. If on conservative treatment, including if necessary transfusions and cortisone, there is, however, no improvement, or relapse follows a remission, splenectomy should be performed.

Remissions are far less common in the adult case and as, in addition, there is a distinct risk of dangerous cerebral hæmorrhage, early splenectomy should be carried out after suitable preparation.

Hypersplenism

It may be accepted that the spleen is capable of destroying blood cells or possibly of preventing their production, maturation, or release in the bone marrow. Hypersplenism describes any state characterized by an excessive activity of this kind, with a consequent significant reduction of cells in the blood. Once again the condition may be idiopathic or secondary to any other cause of splenic enlargement. It is uncertain whether splenic inhibition or splenic phagocytosis is the more important. All cells, red,

SURGICAL INDICATIONS

A. Mechanical. (i) Rupture.
(ii) Penetrating wounds.
(iii) Torsion of the splenic pedicle.
B. Neoplasia. (i) Malignant; Lymphosarcoma.
(ii) Rare benign neoplasms.
C. Cysts. (i) Parasitic; Hydatids.
(ii) True cysts lined by epithelium or endothelium.
(iii) False cysts; Neoplasm with degeneration; Hæmangioma and lymphangioma.
D. Inflammation. Splenic abscess.
E. Aneurysm of the splenic artery.
F. With gastrectomy or œsophago-gastrectomy for carcinoma of the stomach.
G. In the so-called Banti's Syndrome, with or without some form of portal-systemic shunt.

Familial Hæmolytic Anæmia

There is nothing wrong with the spleen in this condition. The fault lies in the patient's red blood cells which, instead of being bi-concave discs, are spherocytes and abnormally fragile. They are destroyed very rapidly by the patient's normal spleen and only by the spleen. Normal red blood cells transfused into the patient are not so destroyed, whilst the patient's abnormal red blood cells transfused into a normal person are rapidly destroyed, unless the latter's spleen has for some reason already been removed, in which case the period of survival is normal. Finally, splenectomy always cures familial hæmolytic anæmia.

The disease tends to run a protracted course with long periods of relatively minor ill health separated by periodic crises of varying severity. These are not due to a significant increase in hæmolysis but to a sudden temporary arrest of red cell production probably due to hypersplenism, for neutropenia is a common accompaniment.

A frequent complication of familial hæmolytic anæmia is cholelithiasis, many small faceted pigment stones forming in the gall bladder.

Treatment. The treatment is splenectomy. In childhood the operation is usually delayed until about the age of ten years unless repeated crises dictate an earlier intervention. In general the patient should be tided over a crisis with blood transfusions and operated upon during a period of remission, but an unusually long or severe crisis occasionally necessitates an emergency splenectomy.

Acquired Hæmolytic Icterus

In this disease there is abnormal sensitization of red blood cells by a circulating antibody. No undue hæmolysis of cells in vitro can be demonstrated, but they become susceptible to phagocytosis in the body to a remarkable degree. Transfused red cells from a normal donor also are rapidly coated with the antibody and sensitized in the same way and rapidly destroyed.

An important investigation in establishing the diagnosis is Coombs' Test. An anti-globulin serum is prepared by immunizing animals against human globulin. Sensitized

Diagnosis rests upon three groups of clinical manifestations:

1. Evidence of violence applied to the region of the spleen.
2. Evidence of loss of blood from the circulation.
3. Evidence of blood in the peritoneal cavity.

Classically, therefore, the patient has sustained an injury to the left lower chest or upper abdomen, where there may be bruising and possibly fractured ribs. After a variable period of time, depending upon the severity of the splenic tear, irritation of the peritoneum by extravasated blood is indicated by abdominal pain, guarding and reflex muscular rigidity, tenderness and rebound tenderness, while loss of blood from the circulation is shown by a raised pulse rate, low blood pressure, pallor, sweating, restlessness, and dyspnœa.

This is the basis of diagnosis but certain additional points are worthy of emphasis:

1. The violence causing rupture is not always severe. Two consecutive children admitted under the writer's care with ruptured spleens each gave a history of trivial violence only.

2. A more obvious injury, such as a fracture or a dislocation, may direct attention away from the abdomen.

3. If the splenic capsule is tough, a blow may rupture the splenic pulp and cause a subcapsular hæmatoma to develop and slowly enlarge. Abnormal physical signs may then be minimal, but splitting of the capsule and sudden torrential hæmorrhage may transform the picture literally in a matter of minutes.

Consideration of these three factors makes it only too clear what a trap may sometimes be laid for the inexperienced or the unwary, particularly by a patient with multiple injuries.

One or two other points of diagnostic significance deserve mention:

1. Loss of blood into the peritoneal cavity must be very considerable before shifting dullness can be noted on examination of the abdomen.

2. Rectal examination may allow detection of blood clot in the pouch of Douglas as well as tenderness of the peritoneum.

3 Auscultation of the abdomen, which should never be omitted in any suspected intra-peritoneal injury, may reveal a silent abdomen, but while inhibition of peristalsis by blood in the peritoneal cavity is common it is not invariable, and normal or exaggerated peristaltic sounds do not exclude a ruptured spleen

4. Irritation of the left leaf of the diaphragm by extravasated blood may give rise to pain referred to the shoulder tip. Sometimes this shoulder-tip pain is suddenly produced by a change in posture, particularly tipping the patient head down. This may be a valuable sign.

5. Plain X-rays of the abdomen are today extensively employed in acute conditions. A large mass of blood clot around a ruptured spleen may depress the splenic flexure of the colon, and a plain X-ray of the abdomen showing the diaphragm separated from this flexure by an opaque effusion is held to suggest a ruptured spleen. The writer has not met with a case diagnosed in this way.

Treatment. The patient's life is in danger from hæmorrhage and in a high proportion of cases the general condition calls for blood transfusion started pre-operatively and continued during operation. At operation splenectomy is performed and injury to adjacent viscera excluded by careful examination. Occasionally, if whole blood is not

white, and platelets may be reduced, the condition resulting being termed splenic pancytopenia, or the white blood cells may be reduced with a normal red cell count and the platelets unaffected or only slightly reduced—splenic neutropenia.

Splenic Pancytopenia

This is characterized by anæmia, neutropenia and thrombocytopenia with a palpable spleen and sternal puncture showing either a normal or hyperplastic marrow. Routine investigation should prevent a diagnosis of idiopathic pancytopenia being made in some more serious condition, such as aleukæmic leukæmia. Hynes (1954) has pointed out that another important diagnostic consideration is identification of a secondary hypersplenism in one of these more lethal illnesses. Occasionally a sudden increase in anæmia may lead to a rapid deterioration in the patient's condition. If this is due to hypersplenism, splenectomy is urgently called for, and if the primary illness is controllable by other measures (e.g. chronic leukæmia) may well retrieve the situation. In splenic pancytopenia splenectomy is likely to be effective in some 80 per cent of idiopathic cases and some 70 per cent of secondary cases.

Splenic Neutropenia

This condition is less common. A low white cell count is accompanied by an increased activity in the marrow with a maturation defect of myelocytes. Intercurrent infections are common, particularly sore throats and ulcers in the mouth. The red cells are within normal limits but some reduction in platelets is usual. Splenectomy is effective in most cases.

A particular variety of splenic neutropenia is the Felty-Sjorgren Syndrome of splenomegaly, neutropenia, and rheumatoid arthritis often with osteoporosis.

"SURGICAL" LESIONS OF THE SPLEEN

Wounds or Rupture of the Spleen

Penetrating wounds of the spleen are rare in civil practice but not uncommon among battle casualties. A splenic injury is nearly always only part of the patient's troubles, coincident damage to the diaphragm, stomach, colon, pancreas, left lung, left kidney, or great vessels being common. Splenectomy is carried out as part of the general exploration and repair of an injury of this kind.

Spontaneous rupture of a normal spleen is rare, but occasionally results from involvement of the splenic vessels in local inflammatory changes in the lesser sac caused by a leaking posterior wall gastric ulcer If the spleen is already the site of advanced disease it may be split or torn by some quite trivial injury; an accident of this kind is naturally uncommon in this country, though it has been described in association with glandular fever.

A spleen of normal size lies wholly under the cover of the left lower ribs and usually the violence causing rupture is fairly severe. The type of violence may be either a direct blow or a crushing injury; thus, street accidents and accidents on the playing fields are common causes.

The result of a laceration of any size is severe intra-peritoneal hæmorrhage and the patient is likely to die of blood loss without surgical aid.

excision advocated by Allison includes also much of the body of the pancreas, the posterior peritoneum of the lesser sac and the loose cellular tissue around the cœliac axis.

The Technique of Splenectomy

Pre-operatively the stomach is emptied by a gastric tube which is left *in situ* during the operation.

General anæsthesia supplemented by an intravenous relaxant is usually employed.

There is no unanimity on the best incision. A left paramedian is adequate in most cases, but a left subcostal incision gives better access to the convexity of the spleen. A transverse incision is less satisfactory. A thoraco-abdominal incision running transversely across the upper abdomen, across the costal arch and up into the eighth intercostal space has been advocated for the very large or very adherent spleen.

After exploration of the abdomen, the next step is mobilization of the spleen by division of the peritoneal reflexions from the diaphragm and left kidney. These avascular folds are put on the stretch and cut through under direct vision with long curved scissors. The spleen can now be lifted into the wound and the vascular attachments dealt with, the pedicle posteriorly and the gastro-splenic ligament with the short gastric vessels anteriorly. Mass ligatures are avoided and vessels individually divided between ligatures. Care should be taken to avoid injury to the tail of the pancreas at the hilum of the spleen. Most surgeons try to secure the splenic artery before the vein in order to return to the circulation the reservoir of blood contained in the organ, and some inject 0·5 ml. of 0·001 per cent adrenaline in order to make the viscus contract.

After splenectomy is performed for a disorder of the blood, search is made for splenunculi, occasionally found in the hilum of the spleen, the transverse mesocolon, or great omentum or retroperitoneally.

It should be possible to secure a dry field at the end of operation and drainage is then unnecessary. If there is any danger of vascular oozing or leakage from a damaged pancreatic tail, however, a corrugated rubber or Penrose drain for a few days must be employed.

Post-operatively the most common complication is a left basal atelectasis, which should be guarded against by routine breathing exercises and forced coughing. Acute dilatation of the stomach is a possible hazard and gastric suction for twenty-four hours with an indwelling tube is generally advisable.

References

Hynes, M (1954) Progress in Clinical Surgery Ed. Rodney Smith. *J & A Churchill Ltd.*, London. p. 178.
Loutit, J. F., and Mollison, P. L. (1946) *J. Path. Bact.* 58, 711

available for transfusion and circulatory collapse is marked, blood from the peritoneal cavity may be filtered and returned into the circulation.

Torsion of the Splenic Pedicle

This is an uncommon condition characterized by recurrent bouts of splenic congestion with perisplenitis and sometimes infarction. In milder cases diagnosis is far from easy Occasionally very severe pain and collapse suggest a perforation or acute pancreatitis.

Treatment is splenectomy. In that they permit rotation to occur, ligamentous attachments are lax and the operation therefore usually not difficult. Occasionally, though, infarction has led to perisplenic adhesions which require division.

Cysts and Neoplasms

Splenic cysts and neoplasms are rarities. Lymphosarcoma is sometimes encountered but usually only as part of a generalized malignant process. Secondary carcinoma very



FIG 135 Neoplasms of the spleen are rare This very large splenic tumour was a hæmangioma.

occasionally occurs Epidermoid, hydatid and angiomatous cysts have been described from time to time and may grow to a large size. The writer removed one hæmangiomatous cyst weighing 8 lb 12 oz (Fig 135)

Aneurysm of the Splenic Artery

This again is a rare condition, occasionally diagnosed as a pulsatile swelling, with a bruit, in the upper abdomen. Calcification in the wall may be visible on X-ray examination. Treatment aims at removing the spleen and aneurysmal vessel together.

Carcinoma of the Stomach

The block excision in cases of operable carcinoma of the upper part of the stomach must include the spleen if adequate clearance of lymph nodes is to be achieved. The

hæmochromatosis. In Africa and the Far East it more often develops as a result of malnutrition or schistosomiasis.

(3) PRE-HEPATIC OBSTRUCTION. Almost invariably this type of portal obstruction is of congenital origin. There may be stenosis or atresia of the portal vein but more commonly it is replaced by a number of thin walled channels which prevent a free flow of blood. This condition is known as congenital cavernomatous portal vein. An important feature of this group of cases is that the liver is normal in appearance and function. The only symptom is bleeding from œsophageal varices which usually comes on in childhood. Without effective treatment an early death from hæmorrhage is certain.

In cases of portal hypertension the venous communications between the portal and systemic systems dilate so that the blood may drain away from the portal system. These communications exist in a number of sites. In the hæmorrhoidal venous plexus the superior rectal vein which drains into the portal system anastomoses freely with the middle and inferior rectal veins which are connected with the systemic system; a small vein which is usually present in the falciform ligament may greatly dilate and carry blood from the portal vein to the parietes in the region of the umbilicus. If the dilation of the veins is of sufficient magnitude it is clinically visible as a caput medusæ. More often the vein is dilated enough to be recognized on portal venography but does not give rise to a caput medusæ (Fig. 137); in the region of the posterior abdominal wall there are communications between the tributaries of the inferior vena cava and the mesenteric veins, and in the submucosa of the stomach and œsophagus there are veins which connect the portal vein on the one hand with veins leaving the œsophagus to drain into the inferior hemiazygos vein on the other. In addition to these anatomical sites of portal-systemic venous anastomoses, vascular adhesions commonly form between the viscera which drain into the portal vein and the parietes. If an operation is performed and adhesions have to be divided some of this flow will be lost. After an operation adhesions to the site of the incision become vascularized to such an extent that a caput medusæ may be formed on the scar. All of these communications are of value to the patient because they result in a lowering of the portal venous pressure. But the veins which dilate in the submucosa of the stomach and œsophagus are a threat to life for as a result of minor trauma or acid erosion an uncontrollable and fatal hæmorrhage may ensue.

Thrombosis of the portal or splenic veins is a complication of portal hypertension. Incomplete or mural thrombosis of the portal vein is not uncommon and increases the difficulty of porta-caval anastomosis.

Pathology of Cirrhosis of the Liver

There has been a marked trend of opinion in recent years towards the belief that cirrhosis of the liver is usually the result of an attack of hepatitis. Milnes Walker has described three types of histological appearance in the liver.

Type 1. There is no abnormality except an increase in the fibrous tissue in the portal tracts.

Type 2. In addition to fibrosis in the region of the portal tracts it spreads in finger-like extensions between the tracts.

These two types are the end results of attacks of hepatitis of varying severity. There is plenty of normal liver parenchyma and liver function tests indicate that there is little

CHAPTER V

THE PATHOLOGY AND TREATMENT OF PORTAL HYPERTENSION

R. E. Horton

During the last decade the surgery of portal venous obstruction has been transformed. Banti's disease and splenic anæmia are obsolete terms which refer only to certain clinical presentations of raised portal vein pressure. Largely as a result of the work of Whipple, Blakemore, Linton and others in America and of Milnes Walker, Learmonth, Hunt and others in this country, the hitherto unhappy lot of these patients has vastly improved. They are no longer observed in medical wards but for many there is a new hope of lasting relief by direct surgery on the portal system having as its object reduction of the portal venous pressure.

Anatomy and Physiology

Blood flows to the liver in the portal vein and hepatic artery. It eventually passes through the liver sinusoids and in these minute vessels the portal vein and hepatic artery blood is mixed. The sinusoids drain into the central veins from which blood is conveyed through the hepatic veins away from the liver. A few anastomoses between the portal vein and hepatic artery are normally present and these give rise to some mixing of portal vein and hepatic artery blood before the sinusoids are reached. In cases in which the flow of blood from the sinusoids is obstructed the hepatic artery pressure causes a rise in the sinusoid pressure and this in turn is reflected in a rise in portal venous pressure. It is for this reason that ligature of the hepatic artery may cause some fall in portal venous pressure in cases of hypertension.

The gross anatomy of the major veins of the portal system is of some importance and differs somewhat from that generally described in textbooks of anatomy (Doutre and Cabanié) The left gastric vein may drain into the splenic or superior mesenteric veins in addition to the portal vein and the inferior mesenteric vein may drain into the superior mesenteric vein as well as into the splenic vein.

Portal Hypertension

When measured with a water manometer and taking the level of the portal vein as a base line the normal portal venous pressure is 100–150 mm. of water. In cases of portal hypertension levels up to 650 mm. of water have been recorded. Hæmorrhage usually occurs in patients in whom the portal pressure is over 300 mm. of water, but this is not always the case.

It is customary to consider the causes of portal hypertension under three headings:

(1) Post-Hepatic Obstruction. This includes such conditions as tricuspid valvular incompetence and constrictive pericarditis which are outside the scope of this article.

(2) Intra-Hepatic Obstruction. This is due to cirrhosis. In the United Kingdom it is usually the result of an attack of hepatitis but it may rarely occur in cases of

Other patients present with the symptoms of cirrhosis and on examination are found to have splenomegaly or there may be ascites and splenomegaly. In young adolescents and children who present themselves with hæmatemesis and splenomegaly it is likely that the portal hypertension is due to congenital cavernomatous portal vein rather than to cirrhosis.

The presence of œsophageal varices may be confirmed by barium swallow (Fig. 136) or by œsophagoscopy and if there are no œsophageal varices it may be confidently stated that the portal pressure is not elevated.

The blood is examined to detect the presence of anæmia or signs of hypersplenism and the function of the liver is assessed. The serum proteins are of particular importance and a serum albumen level of less than 3 gm. per cent is of serious import; one below 2·5 gm. per cent is an absolute contra-indication to a porta-caval shunt. If the level is between 2·5 and 3 gm. per cent the danger of a major operation is considerable and the risk of further and fatal hæmorrhage must be carefully assessed. The serum alkaline phosphatase is generally elevated in cases of obstructive jaundice and normal in cases of hepatic jaundice. In cases of cirrhosis elevation of the level of alkaline phosphatase is an indication of very gross liver damage. The flocculation tests such as the thymol turbidity test are positive in cases of hepatitis and normal values are obtained in cases of obstructive jaundice. The flocculation tests give some idea of liver function and are of value with the serum protein estimations in assessing the progress of hepatitis. In some cases of subacute hepatitis it may be advisable to watch the course of events for a time and it is here that the flocculation tests are of especial value.

The serum proteins, serum alkaline phosphatase and flocculation tests are reviewed together in all cases and an opinion on the liver function is based on them.



FIG 136 Barium swallow to show œsophageal varices

Peritoneoscopy

In doubtful cases of hepato- and splenomegaly peritoneoscopy may disclose that the liver is cirrhotic. In cases of enlargement of the liver of doubtful cause peritoneoscopy may easily resolve the problem The operation is performed under local anæsthesia and the instrument inserted in the midline below the umbilicus. When the peritoneal cavity

disturbance of hepatic physiology; portal vein obstruction is variable but may be severe. In the past these relatively insignificant amounts of fibrosis have often been referred to as early cirrhosis. The inference that the process is a progressive one is incorrect for the condition may be of very long standing as may be apparent from the history. There is no evidence that the liver pathology in these two types is progressive. A further attack of hepatitis alone can give rise to an increase in the liver fibrosis.

In *Type* 3 there is a much more serious disturbance of the liver which has been disorganized by an attack of hepatic necrosis. In these cases there may be considerable elevation of portal venous pressure but the clinical picture is dominated by the signs of impaired liver function and ascites is commonly present. The clinical course is a more rapid one and the outcome fatal.

In Types 1 and 2 the histology in the liver is the result of a process of organization which follows hepatitis and when it is complete no further changes occur. But in Type 3 there is such extensive scarring that the central veins are obstructed in addition to the portal veins. There is congestion of the liver sinusoids and progressive failure of hepatic function.

Clinical Aspects

In this country most cases of intra-hepatic obstruction due to cirrhosis result from an attack of hepatitis. It is often possible to elicit a history of jaundice but in other cases in which there is no such history there may have been a subicteric attack of hepatitis. In all cases of portal hypertension enlargement of the venous anastomosis between the portal and systemic venous systems takes place. The enlarged veins in the œsophagus and stomach are a potential source of danger and may give rise to severe attacks of hæmorrhage. It is rare for such a hæmorrhage to be fatal on the first occasion but once severe bleeding has taken place a fatal outcome from hæmorrhage within the next few years is likely. The development of collaterals elsewhere may so lower the portal pressure that the interval between episodes of hæmorrhage may rarely be as much as 10 years or more. Enlargement of the porta-systemic connections in the anal canal gives rise to hæmorrhoids and the symptoms occasioned by these may be prominent. Although hæmorrhoids are a surprisingly rare manifestation of portal hypertension the recognition that hæmorrhoids may be due to this cause is important Failure results not only in lack of treatment of portal hypertension but in early recurrence of the hæmorrhoids. In other cases the dilation of one or more veins in the falciform ligament promotes a free connection between the portal system and the veins in the anterior abdominal wall and the latter may be visible and greatly dilated

In cases of portal hypertension due to cirrhosis signs of liver failure may dominate the clinical picture if the histology is of Type 3. The complexion is sallow and the face marked by capillary telangiectasia. Ascites may be present and in these cases the serum albumen is nearly always low The cause of ascites in cirrhosis has not been clearly established, but it is certainly related to impaired liver function and never occurs in patients with a pre-hepatic cause of hypertension such as congenital cavernomatous portal vein in which the liver is normal

When portal hypertension is present the congested spleen enlarges and in a few cases the clinical syndrome of hypersplenism may be encountered and there may be leucopenia and thrombocytopenia.

The radiographs are also useful in showing the disposition of large collateral vessels and the main source of the veins taking blood to the varices.

It is also possible by this technique to determine the patency of a porta-caval shunt.

Treatment of Portal Hypertension

When Banti's disease and splenic anæmia were common diagnoses the operations of splenectomy and omentopexy were occasionally performed. Splenectomy is now thought to be contra-indicated as spreading thrombosis along the splenic vein will make subsequent surgery on the veins difficult or impossible. Howells showed in 1938 that splenectomy influenced neither the expectation of life nor incidence of hæmatemesis and this experience has been repeatedly confirmed, and in fact by dividing the vascular adhesions which connect the spleen to the parietes the operation may contribute to a further rise in portal pressure. Omentopexy, which was designed to try to encourage the formation of natural connections between the portal and systemic systems, has also been abandoned as it has little effect on the portal pressure or ultimate prognosis.

The principles on which modern surgery is based are that in many cases the liver pathology is not progressive and that the patient is likely to die of hæmorrhage. If the portal pressure can be effectively lowered the result will not be prejudiced by advancing liver failure. It has been found that the most satisfactory means of reducing portal pressure is by means of an end-to-side anastomosis between the portal vein and the inferior vena cava. It is essential that the shunt be a wide one and in most cases it is possible to get a lumen of about 1 cm. With such a shunt the chance of its subsequent closure by thrombosis is unlikely and further bleeding from œsophageal varices is rare. If the portal vein is unsuitable for a shunt operation it may be possible to do a splenorenal anastomosis.

Rienhoff introduced the operation of hepatic artery ligation and this operation has recently received some additional support from McFadzean and Cook. The operation has the advantage of being technically much easier than a porta-caval shunt and theoretically it should help to lower the portal venous pressure. In practice the effect is disappointing and the operation has the additional drawback of causing liver necrosis and death in a small proportion of cases.

A number of patients remain in whom there is no contra-indication to major surgery but in whom shunt operations cannot be done, for anatomical reasons. These include patients with congenital cavernomatous transformation of the portal vein and those with extensive mural thrombus. Here it is not possible to lower the portal venous pressure and the surgery is directed to the varicosities themselves.

Porta-caval Anastomosis

This operation is indicated in patients in whom there has been severe bleeding from gastric or œsophageal varices or in whom there is serious risk of such a hæmorrhage because of the presence of varices. The operation may only be recommended if portal venography has shown the presence of a suitable portal vein and the liver function tests are satisfactory. The flocculation tests must show minimal or no flocculation and the serum proteins must be nearly normal. The operation is dangerous when the serum albumen is less than 3 gm. per cent and likely to be fatal if it is less than 2·5 gm. per cent. Blakemore has recommended this operation in cases in which the main symptom

has been filled with air it is possible to see the liver, stomach, much of the peritoneum and, with some manœuvring, the edge of the spleen. The advantage over laparotomy is a clear one for it is possible to discharge the patient the following day if the case is not one of cirrhosis or no further treatment is entertained.

Portal Venography

This recent addition to the investigations of portal hypertension has proved to be very useful. It was first described by Albeatici and Campi and has recently been reviewed by Walker, Middlemiss, and Nanson. The investigation is a simple one and without



(Professor R. Milnes Walker's Case)

FIG. 137 FIG. 138

PORTAL VENOGRAM. FIG. 137 shows the deposit of dye in the spleen and the characteristic course of the splenic and portal veins. The portal vein runs obliquely to the right and dye fills the hepatic branches. In Fig. 138 a dilated tortuous vein is seen running from the right branch of the portal vein towards the midline. At operation this vein was found to be in the falciform ligament.

hazard in patients with splenomegaly. Premedication with Omnopon is followed with 100 mgm. of Pethidine intravenously just before the splenic puncture. The needle of S.W.G. 17 is inserted through the estimated depth of the parietes under local anæsthesia. It is best directed to the spleen about 4–5 cm. behind the costal margin in the posterior axillary line and it usually traverses the 10th intercostal space. The patient is instructed to take a deep breath and to hold it and the needle is then inserted a further 2 cm. into the splenic substance and the injection of 30 ml. of 70 per cent Diodrast is made as quickly as possible. Two radiographs are taken, the first at completion of the injection and the other 2 seconds later. It is important for the patient to stop breathing while the needle is in the spleen.

The radiographs give a clear picture of the portal vein and it is possible to know whether the vein will be suitable for an anastomosis or not. Useless operations of exploration may be avoided and if the portal vein is cavernomatous or greatly reduced in calibre by mural thrombosis operations other than those directed to the portal vein itself will have to be elected.

glutamate in 80 ml. of water is added to 500 ml. of 5 per cent glucose and given intravenously in 3–4 hours. As an alternative, glutamic acid may be given orally in 0·5 gm. tablets to a total of 20 gm. in a day.

Patency of the shunt may be confirmed by portal venography provided the spleen has not been removed. So long as the shunt remains patent and there is no reason to believe that late thrombosis will occur, the risk of hæmorrhage is greatly reduced. The spleen becomes rapidly smaller and the œsophageal varices less tense. But the latter do not disappear and in a few cases further bleeding has taken place in spite of an adequate porta-systemic shunt. If this happens a direct attack on the varices is indicated without delay for such a hæmorrhage may be fatal.

Spleno-renal Anastomosis

This operation is indicated when there is portal hypertension which cannot be treated by porta-caval anastomosis because of the condition of the portal vein. It is more difficult than the porta-caval anastomosis and the resulting shunt is much more liable to thrombose because of its smaller calibre. It is an end-to-side anastomosis with splenectomy. The end-to-end operation which also involved nephrectomy is now obsolete.

The patient is positioned at 45 degrees with the left side turned upper-most and the approach is made through the bed of the ninth rib with the incision extending 3–4 in. across the costal margin. The lesser sac is entered through the gastro-colic omentum and the splenic artery ligated in continuity. This step is always valuable as a preliminary to splenectomy as it results in shrinkage of the spleen. The artery is located where it makes a loop above the upper border of the pancreas and it is ligated with thread. Attention is then turned to the pedicle of the spleen after division of the peritoneum along its outer border. The vascular pedicle is dissected with great care and every effort is made not to damage the splenic vein which is thin walled and friable. The splenic vein is controlled with a bull dog clamp and the spleen removed. The retroperitoneal dissection to isolate the renal vein is then proceeded with and the latter is carefully isolated and its proximal end controlled with a Blalock clamp. The distal tributaries are controlled by encircling ligatures. An anastomosis is then made between the splenic and renal veins using a continuous everting mattress stitch. It is important that the veins should be mobilized enough to make the anastomosis without tension and also that it should be at least 1 cm. diameter. If such a diameter cannot be attained thrombosis is very likely and the operation should not be proceeded with. But in cases of portal hypertension the splenic vein is usually considerably dilated and a shunt of this diameter is generally possible. The thoraco-abdominal wound is closed in the usual way with drainage to the abdomen for about 48 hours.

The complications which follow spleno-renal anastomosis are largely the same as those of porta-caval shunt. If a patient with poor liver function has been selected for operation he may go into hepatic coma. The danger of thrombosis of the anastomosis is greater than after porta-caval shunt and this is due to a number of factors. The stoma is placed further from the site of obstruction than in the case of a porta-caval shunt and the flow of blood in the splenic vein is reversed after a successful shunt. In addition, thrombosis is made more likely by splenectomy which is followed by an increase in the platelet count and in increased susceptibility to intravascular coagulation. A liberal fluid and glucose intake helps to protect the liver after porta-systemic shunt operations.

is ascites. He suggests that improvement in liver function follows a shunt which reduces the portal pressure. But the opinion in this country is that in patients with ascites the operation is contra-indicated because of the impaired function which it implies. When the ascites cannot be controlled with a high protein and salt free diet the prognosis is very bad.

Blakemore still employs an upper abdominal approach, but much better access is obtained through a right thoraco-abdominal incision and this is more commonly employed in this country.

The pre-operative preparation is the same as for any thoracotomy. The patient should have had breathing exercises started and begin penicillin on the day before the operation.

Two pints of blood should be available as there is always some loss of blood from small collateral veins which are divided in the approach to the portal vein.

The operation is a thoraco-abdominal one and the patient is positioned at 45 degrees with the right side uppermost. The incision extends along the length of the ninth rib and for some 5 cm. beyond the costal margin in the same line. The ninth rib is resected and its cartilage split longitudinally to facilitate sewing up. The pleural and peritoneal cavities are opened and the diaphragm split to the coronary ligament of the liver. Retractors are inserted and the liver displaced upwards. The peritoneum between the liver and kidney is incised and the incision carried along the line of the inferior vena cava. The foramen of Winslow is located and the peritoneum is incised along its lower and anterior borders. Dissection proceeds through this peritoneal incision until the portal vein is identified; a number of small collateral veins are divided at this stage. If possible the two main branches of the portal vein are identified and ligated separately. Ligature of the branches individually has the advantage of safety for the junction prevents the ligatures from slipping off. The portal vein is controlled with a Blalock clamp and divided just below its point of division After clearing some 4–5 cm. of the anterior surface of the inferior vena cava a part of its wall is picked up in a Brock mitral clamp. An opening is made in this part of the inferior vena cava to correspond to the end of the portal vein and the two are anastomosed by a continuous everting mattress suture. By using the Brock clamp in this way to isolate a part of the vena cava for anastomosis, the main flow in the cava is uninterrupted during the time that the anastomosis is being made. Other suture techniques have been found less satisfactory and the use of a vitallium tube certainly adds to the risk of thrombosis The portal pressure should be taken before and after the shunt has been completed and a wedge of liver is removed for histological examination. The wound is closed with a drain to the pouch of Rutherford Morison. It is not necessary to drain the pleural cavity and no special post-operative treatment is necessary except that generally given after thoracotomy. Post-operative heparin is not used.

Complications of the porta-caval shunt operation are uncommon. They comprise those generally met after abdominal or thoracic operations But if patients with advanced impairment of liver function are operated on there may be dangerous complications. If the portal blood is diverted from a liver of profoundly disturbed function the patient may be precipitated into a condition of hepatic coma the termination of which is likely to be fatal. In this condition the patient may recover from the anæsthetic and lapse into unconsciousness some 24 hours later The exact mechanism of hepatic coma is unknown. Walshe has recently shown that intravenous glutamic acid has a very beneficial effect on the course of the disease and may restore consciousness and save life. 23 gm. of sodium

glutamate in 80 ml. of water is added to 500 ml. of 5 per cent glucose and given intravenously in 3–4 hours. As an alternative, glutamic acid may be given orally in 0·5 gm. tablets to a total of 20 gm. in a day.

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The complications which follow spleno-renal anastomosis are largely the same as those of porta-caval shunt. If a patient with poor liver function has been selected for operation he may go into hepatic coma. The danger of thrombosis of the anastomosis is greater than after porta-caval shunt and this is due to a number of factors. The stoma is placed further from the site of obstruction than in the case of a porta-caval shunt and the flow of blood in the splenic vein is reversed after a successful shunt. In addition, thrombosis is made more likely by splenectomy which is followed by an increase in the platelet count and in increased susceptibility to intravascular coagulation. A liberal fluid and glucose intake helps to protect the liver after porta-systemic shunt operations.

Direct Operations on the Varices

These operations are indicated for cases in which bleeding from varices has taken place, but in which the porta-systemic shunt operations are impossible. It includes the group of patients with congenital cavernomatous portal vein and also those with portal hypertension due to cirrhosis in whom the vein is the subject of extensive mural thrombosis. It is also indicated in patients who continue to have hæmorrhage after a splenorenal shunt and in whom thrombosis of the anastomosis may have occurred. Rarely it will be found that the varices still bleed after a porta-caval shunt in spite of patency of the anastomosis and again an operation on the varices is indicated.

A variety of methods have been employed to deal with the varices. Endoscopic injection with sclerosing fluid has been recommended but has not become popular and is probably not very effective.

The operation of œsophageal transection has been described by Milnes Walker. In this operation the œsophagus is approached through a left lower thoracotomy. It is isolated and controlled with intestinal clamps Effective interruption of the veins is achieved by dividing the œsophagus completely and resuturing it. Although it is almost certain that veins will grow across the scar in due course, time is given in which a more free collateral circulation can be established. Various modifications of this operation have been described, and Boerema advocates one in which the œsophagus is opened longitudinally and the varices are then under-run with sutures. All these operations on the œsophagus have the disadvantage that they may be followed by stricture which will require repeated bouginage. In addition, further hæmorrhage may take place from the varices in the stomach and œsophagus below the level of transaction.

Tanner's operation of gastric transection makes an equally effective interruption between the portal and systemic venous systems without the disadvantages of the operation of œsophageal section. In this operation the portal and systemic venous systems are disconnected by an operation in which the stomach is transected and resutured somewhere below the cardia. In addition the vasa brevia and the left gastric pedicle are ligated sparing only the ascending branch of the left gastric artery to preserve a blood supply to the cardiac end of the stomach. Tanner recommended that this operation should be performed through an upper abdominal approach and he suggested that it could be used as an interval operation or as an emergency procedure for hæmorrhage. Access to the upper third of the stomach may be difficult in cases of portal hypertension because of vascular adhesions in the region of the vasa brevia and a better approach can be obtained by doing the operation through a left lower thoracotomy and dividing the diaphragm.

Phemister and Humphreys (1947) introduced the operation of œsophago-gastrectomy removing the lower œsophagus and upper third of the stomach and reconstituting the bowel by œsophago-gastrostomy. This operation has the effect of excising the area bearing varices It also interrupts the portal and systemic circulations, but it destroys the cardiac sphincter of the stomach and has the disadvantage that it may be followed by acid regurgitation into the œsophagus with all its train of serious sequalæ. Learmonth (Macpherson) who has practised this operation more recently, claims that digestive disturbances are less marked than in cases in which the operation is performed for malignant disease, but the operation has probably little advantage over the lesser procedure of gastric transection

Ligature of the Hepatic Artery. Rienhoff originally performed this operation which is designed to reduce portal venous pressure. It is based on the assumption that in cases of intra-hepatic obstruction the raised portal venous pressure is maintained by hepatic artery pressure through the sinusoids in which the circulations are connected and also through the increased anastomosis which are present between the portal vein and hepatic artery. There is some evidence that ligature of the hepatic artery results in a lowering of portal venous pressure, but it is a dangerous operation which may be followed by massive hepatic necrosis and death. McFadzean and Cooke who have reported their experiences of the operation had 3 such cases in a series of 15 in which hepatic artery ligation was performed. Some confusion exists because of the variable origin of the blood supply of the liver and in cases in which the patient has survived hepatic artery ligature there may have been an accessory supply direct from the gastro-duodenal or left gastric arteries.

Clearly the operation is a dangerous one and its place in the surgery of portal hypertension is doubtful. It may be indicated in patients with a good portal vein in whom porta-caval shunt is contraindicated because of impaired liver function for it is technically a much easier procedure. It is contra-indicated in cases of extra-hepatic obstruction to the portal vein for in these cases ligature of the hepatic artery is certain to be followed by liver necrosis.

Treatment of Œsophageal Hæmorrhage. Although there are some advocates of immediate operation on the lower œsophagus to stop free bleeding the usual course adopted is a conservative one. Death is not common in the first attack but bleeding from varices is always very serious. The patient is kept at absolute rest and if the systolic blood pressure falls below 100 mm. Hg a blood transfusion is given. It is important to give blood judiciously as if the blood pressure is raised too abruptly the bleeding will be exacerbated. Rowntree has recommended the use of an inflatable balloon in the lower œsophagus, but the method does not appear to be of great practical value.

Results

The magnitude of the benefit which patients with portal hypertension derive from porta-caval anastomosis may be judged from the results of Blakemore who has the largest experience of the operation. His operative mortality for porta-systemic shunts is 18 per cent in a total of 166 cases The rather high figure may be accounted for by the fact that he includes ascites as an indication for the operation. In Milnes Walker's series in which the indication for the operation was hæmorrhage and cases with serious impairment of liver function were excluded, there have been only 2 deaths in the last 30 cases Both of these had a moderate degree of impairment of liver function.

Blakemore, who has followed his cases for up to 7 years, has a survival rate of 95 per cent in extra-hepatic obstruction and 82 per cent in intra-hepatic obstruction. Only 2 patients on whom a porta-caval shunt had been performed subsequently died of gastro-intestinal hæmorrhage. Further hæmorrhage may be due to closure of the anastomosis, but it occasionally happens after a porta-caval shunt although the anastomosis is patent. This is a tremendous benefit for observations on the natural history of cirrhosis of the liver indicate that 50 per cent to 80 per cent of patients are dead within a year of the first hæmorrhage.

In Blakemore's series there were 4 deaths from liver failure after a shunt operation.

With increasing experience this complication should be rare for it usually indicates an error of selection for operation.

Operations on the varices which are performed when porta-systemic surgery is not applicable are less likely to be of permanent value. It is too early to judge these operations finally but they prevent further bleeding from residual varices.

References

Albeatici, S. and Campi, L. (1951) *Minerva Med Roma*. 42, 593
Blakemore, A. H. (1952) S G. and O. 94, 443.
Boerema, I (1949) *Arch. Chir. Neerland.* 1, 253.
Doutre, L P and Cabanié, H. (1952) *Presse Med.* 60, 1129.
Howells, L. (1938) *Lancet*, 1, 1320.
Linton, R. R. and Warren, R. (1953) *Surgery*, 33, 243.
McFadzean, A J. S and Cook, J. (1953) *Lancet*, 1, 615.
MacPherson, A. I S (1953) *Edin. Med. J.* 60, 13.
Phemister, D. B and Humphreys, E. M (1947) *Ann Surg*. 126, 397.
Rienhoff, W F. (1951) *Bull. Johns Hopkins Hosp.* 88, 368.
Rowntree, C G 1(947) *J.A.M A* 135, 630.
Tanner, N C. (1950) *Proc. R.S M* 43, 147.
Walker, R. M. (1952) *Lancet*, 1, 729.
Walker, R. M., Middlemiss, J H. and Nanson, E. M. (1953) *B.J.S.* 40, 392.
Walshe, J. M. (1953) *Lancet*, 1, 1075.

CHAPTER VI

PERITONEOSCOPY

JOHN HOSFORD

PERITONEOSCOPY, the viewing of the peritoneal cavity through an instrument introduced through the abdominal wall is a simple but comparatively little used method of examination.

HISTORY

Ott, a Russian, in 1901 seems to have been the first to look into the peritoneal cavity through a small opening: he used a speculum and head mirror and it is not known how much he saw.

The first report of an attempt at peritoneoscopy by an instrument carrying its own light was by Kelling in Germany in 1910: he used a cystoscope and had a fair measure of success.

Various other attempts with cystoscopes, sigmoidoscopes, etc., were briefly reported including one in 1925 by Rendle Short who coined the word "cœlioscopy" and used a cystoscope. It was not, however, until Ruddock of Los Angeles in 1934 published his first paper describing his own peritoneoscope and its use that a wider interest was taken in the subject. Since then there have been many articles in the American Medical literature on the subject though only a few in this country. Of the latter the first was by Milnes Walker of Bristol in 1942 who gave a full account of the subject using a Ruddock's peritoneoscope. Peritoneoscopy was the subject of a meeting of the Royal Society of Medicine in 1943 and in addition to Milnes Walker as opening speaker was Cooke of Oxford who clings to the name of laparoscopy.

In spite, however, of the fact that a sound technique has been established for peritoneoscopy it remains a singular fact that it is still only used comparatively seldom in this country and only a few people have troubled to make themselves familiar with it.

INDICATIONS

While a good view may be obtained of much of the abdomen by peritoneoscopy, in practice it is found that it is most often of help in the diagnosis of liver conditions, e.g. in determining whether a swelling in the upper abdomen is arising from the liver or not, in seeing metastases in the liver when the latter is not palpable or distinguishing metastases from cirrhosis when it is palpable, etc.

Peritoneoscopy is helpful with any doubtful abdominal lump provided that the physical signs suggest that it is in the anterior part of the abdomen: it is not likely to be of value in the case of retroperitoneal swellings.

The cause of a doubtful ascites is frequently made clear by peritoneoscopy—multiple malignant nodules or tubercles on the peritoneum or omentum, or dilatation and congestion of veins are easily seen.

With increasing experience this complication should be rare for it usually indicates an error of selection for operation.

Operations on the varices which are performed when porta-systemic surgery is not applicable are less likely to be of permanent value. It is too early to judge these operations finally but they prevent further bleeding from residual varices.

References

Albeatici, S. and Campi, L. (1951) *Minerva Med Roma.* 42, 593
Blakemore, A. H. (1952) S. G. and O 94, 443.
Boerema, I. (1949) *Arch. Chir. Neerland.* 1, 253.
Doutre, L. P. and Cabanié, H. (1952) *Presse Med.* 60, 1129.
Howells, L. (1938) *Lancet*, 1, 1320.
Linton, R. R. and Warren, R. (1953) *Surgery*, 33, 243.
McFadzean, A. J. S. and Cook, J. (1953) *Lancet*, 1, 615.
MacPherson, A. I. S. (1953) *Edin Med J.* 60, 13.
Phemister, D B and Humphreys, E. M (1947) *Ann. Surg.* 126, 397.
Rienhoff, W F. (1951) *Bull Johns Hopkins Hosp.* 88, 368.
Rowntree, C G 1(947) *J.A.M.A* 135, 630
Tanner, N. C (1950) *Proc R.S M* 43, 147.
Walker, R M (1952) *Lancet*, 1, 729.
Walker, R. M., Middlemiss, J. H and Nanson, E. M. (1953) *B.J.S.* 40, 392.
Walshe, J. M. (1953) *Lancet*, 1, 1075.

examination is finished as much air as possible is let out through the sheath, which is then withdrawn, and the tiny opening is closed. Unless the patient is stout it is usually possible to put one stitch in the peritoneum, and the skin is closed with a stitch and sealed with mastisol.

The information that can be derived from peritoneoscopy is not, of course, by a considerable measure so great as that which can be obtained from a laparotomy because only the anterior surface of the structures in the anterior part of the abdomen can be seen. However, by the simple manœuvre of inserting a slim metal rod through a separate

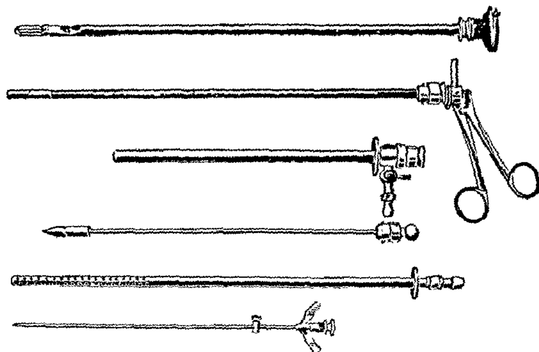


Fig 139

Forobhque Telescope.
Biopsy Forceps
Peritoneoscope Sheath.
Peritoneoscope Trocar
Perforated Suction Tube
Trocar and cannula for pneumoperitoneum

puncture hole in the abdominal wall the edge of various structures can be raised and a view obtained under them: the liver edge can be raised and more of the stomach or gall bladder be seen. the omentum can be raised to expose the underlying intestine.

Ascites is no contra-indication to peritoneoscopy The sheath of the instrument is introduced in the ordinary way and after withdrawing the trocar a perforated suction tube is passed and the peritoneal cavity emptied of fluid. The author has never known the removal of many pints do harm though in a very tense ascites of long standing it might be wise to let off some of the fluid slowly over a longer period If the fluid is not drawn off the view is likely to be confused either from looking at organs from partly under water or looking through froth.

By using the special biopsy forceps made for the purpose small bits of tissue can be obtained through the peritoneoscopy sheath. A small portion of tissue may be removed from any organ or tumour though it is mainly of use in connection with liver biopsies

The whole technique of peritoneoscopy is not difficult to learn and complications

If necessary a chronically inflamed gall bladder can be recognized by peritoneoscopy though the presence of small calculi in a fairly normal thin-walled gall bladder cannot be determined

It is difficult to see the spleen unless it is at least moderately enlarged.

There is little chance of gaining much help in the diagnosis of doubtful peptic ulcers or early carcinoma of the stomach. In either acute or "chronic" appendicitis peritoneoscopy is not indicated.

Uterine and ovarian tumours can usually be well seen with the patient in the Trendelenburg position but peritoneoscopy is not often used because a diagnosis is made by other means.

A carcinoma of the anterior surface of the stomach can be well seen and valuable help may be obtained by seeing growth which has spread to parietal peritoneum, omentum, etc.

TECHNIQUE

Peritoneoscopy should be carried out in an operating theatre with full aseptic ritual and it is of much convenience to have a table which tilts into the Trendelenburg and reverse Trendelenburg positions.

The instrument (Fig. 139) consists of:

(1) A trocar and cannula for producing a pneumoperitoneum.

(2) The outside sheath of the peritoneoscope.

(3) A trocar for introducing the sheath.

(4) The telescope.

The procedure consists in distending the abdominal cavity with air introduced through the cannula in the mid-line a little distance above the pubes. This lifts the abdominal wall away from the intestines and omentum, and at the same time makes the abdominal wall firm because the air is pumped in by an ordinary rubber bellows with a valve until a fair degree of pressure is reached.

An incision about ¼ in (0·6 cm.) in length is then made through the skin a little distance below the umbilicus in the mid-line, and the peritoneoscope, held very firmly, is sharply but very carefully pushed through into the peritoneal cavity. The introducing trocar of the peritoneoscope is then withdrawn from the sheath, and the telescope which carries its own light, is passed down the sheath, into which it fits with an air-tight washer. Air which has escaped will be replaced so as to give room in which the instrument may be moved about and the examination of the cavity then proceeds.

The instrument is inserted below the umbilicus because this makes it easy to see both sides of the upper abdomen by passing it upwards first on one side of the falciform ligament and then on the other Adhesions may cause trouble in moving the instrument about. It is dangerous to insert the instrument into the abdomen close to a previous incision as intestine may be adherent to the abdominal wall at this point and be injured.

By tilting the patient with the head up a better view can be obtained of the anterior surface of the liver and stomach Similarly a steep Trendelenburg tilt will help to see the pelvis though it is sometimes difficult to get all the small gut up out of the way.

Under local anæsthesia many patients do not complain at all, though others resent the abdominal distension with air, and a few experience pain when the instrument rubs against the parietal peritoneum as it is moved about inside the abdomen. When the

and troubles are few. The view obtained is good and much easier to understand and interpret than is the case with cystoscopy and gastroscopy.

The two obvious dangers are perforation of some viscera by the instrument and hæmorrhage. The author has fortunately met neither of these. The moment of greatest danger is as the instrument with its pointed trocar in position is pushed through the abdominal wall: it may impale a loop of gut against the vertebræ or transfix the great vessels as they lie on them: but the intestines are slippery things and readily move out of the way and if the instrument is held firmly it should not reach the posterior abdominal wall.

In a few patients an area of surgical emphysema occurs around the little opening in the abdominal wall due to escape of more air from the peritoneal cavity after the single stitch has been put in the skin. It is slight, quickly disappears and has never been known to do harm.

A more troublesome though not really serious complication may occur when introducing the preliminary trocar and cannula for the pneumoperitoneum. Care must be taken to make sure that it has gone right through the parietal peritoneum as well as the linea alba. Sometimes the peritoneum is rather loosely attached to the abdominal wall and it gets pushed ahead of the cannula instead of pierced by it with the result that the air when pumped in strips the peritoneum up over a very wide area causing an internal surgical emphysema. This state of affairs is difficult to correct and makes examination most awkward or even impossible.

Great care must be taken when doing biopsies with the special instrument that bleeding does not continue.

References

Cooke, A. M (1943) *Proc. Roy Soc. Med* 36, 448.
Kelling, G. (1910) *Munch. Med Wochr.*, 49, 21.
Milnes Walker, R. (1942) *Lancet* (i) 159.
Ott, D. (1901) *J. Akush. i Zhensk. Bolez*, 15, 1045.
Rendle Short (1925) *Brit. Med. J.* (2) 254.
Ruddock, J. C. (1934) *West. J Surg* 42, 392.
Ruddock, J C. (1937) *Surg Gynec. Obstet* 65, 623
Ruddock, J. C (1939) *Sth Surg.* 8, 113

CHAPTER VII

ACUTE INTESTINAL OBSTRUCTION

RODNEY SMITH

ACUTE intestinal obstruction is one of the commonest of emergencies encountered in surgery and, at any rate until quite recently, one which carried a higher mortality than appeared reasonable, having regard to the apparent simplicity of the problem. Of recent years, however, the results of treatment have improved steadily and the reason for this satisfactory state of affairs is undoubtedly a fuller understanding of pathology and a more accurate assessment of the lethal factors involved. It is, therefore, right to devote a certain space to a discussion of pathology and the whole subject will be presented under the headings already employed in another work (Rodney Smith, 1947) reference to which is acknowledged:

(1) Varieties and causes of acute intestinal obstruction.
(2) Pathology.
(3) Diagnosis.
(4) General lines of treatment.
(5) Individual examples of acute intestinal obstruction.

VARIETIES AND CAUSES OF ACUTE INTESTINAL OBSTRUCTION

Acute obstructions are traditionally divided into two main groups:

(*a*) *Mechanical or organic obstructions,* where the onward passage of intestinal contents is arrested by some clearly demonstrable physical barrier.

(*b*) *Functional obstruction,* where the onward passage of intestinal contents ceases on account of some derangement of peristalsis.

Mechanical obstruction is the commoner of the two. The *cause* may lie outside the bowel, such as an intraperitoneal band or the neck of a hernial sac, in the bowel wall, such as a stenosing carcinoma, or in the bowel lumen, such as an impacted gall stone. Certain well recognized *types* of organic obstruction exist which are quite distinct, although in an individual case one type may pass into another. These are:

Simple occlusion.
Closed loop obstruction
Intestinal strangulation

and these may affect the small or the large intestine.

Functional obstructions similarly fall into two groups, paralytic and spastic, of which the latter is rare.

Pathology

Simple Occlusion. Simple occlusion of the bowel brings about an orderly sequence of easily understood changes. Below the site of occlusion the bowel empties and remains contracted and empty. Above, the bowel fills up and becomes progressively dilated with

liquid and gas. Liquid, apart from a small amount of swallowed fluid, is derived from the digestive juices secreted by the stomach, duodenum, pancreas, liver, and small bowel. Gas comes mainly from swallowed air. As the bowel becomes dilated the intra-luminal tension rises. This rise in tension is directly responsible for the other essential changes caused by occlusion. Peristalsis is a reflex mediated by the myenteric plexus of Auerbach, and evoked by a stimulation of the nerve endings in the wall of the bowel. A rising intra-luminal tension provides a very effective stimulus. The response is first increased peristalsis, then more violent and inco-ordinated contractions and possibly waves of reversed peristalsis, and finally, when gross stretching has occurred, inhibition of peristalsis and passive dilatation. This ultimate inhibition of peristalsis by increasing stretching of the bowel wall may be ascribed either to a direct effect upon intestinal musculature, which is in accord with accepted physiological principles, or to the indirect effect of interference with the blood supply. It is probable that both effects are of importance In any event the rise in tension eventually leads to circulatory stasis, œdema and partial or complete paresis.

The Secretion and Absorption of Fluid. As the tension rises in the occluded intestine, there is an increase in the amount of fluid secreted by it. The cause is wholly, or at least mainly, mechanical. Compression of the venous side of the local circulation occurs early, and with it occurs a rise in the capillary pressure, slowing of the local circulation and anoxic damage to the capillary bed The outpouring of fluid into the lumen of the bowel is accompanied by a similar outpouring of fluid into the intercellular spaces with the production of local œdema. The capacity of the intestine to absorb fluid coincidentally falls with the slowing local circulation and a rapid accumulation above the site of occlusion results

Gas in the Bowel. Apart from swallowed air, a rise in the bacterial content of the bowel is reflected in the increased fermentation and production of gas, while another source is the diffusion of gases into the lumen of the bowel from the blood stream.

Organisms in the Bowel. The rise in the bacterial content is accompanied by a change in the nature of the organisms themselves, proteolytic flora tending to flourish at the expense of other types and gram-negative organisms outgrowing gram-positive. This change is of some significance in cases of peritonitis associated with intestinal obstruction.

The Blood Vessels in the Bowel Wall. As the tension rises in the obstructed bowel, important effects are produced by compression of the blood vessels in the bowel wall. A slight rise in tension causes venous stasis with a rise in the capillary pressure, reflected by increased capillary permeation and transudation of a blood-stained fluid into the lumen and the intercellular spaces of the intestine A further rise in tension still further slows the circulation until a point is reached at which the viability of the bowel is jeopardized. It is probable that by the time that this stage is reached the effects of pressure are no longer confined to the venous side of the circulation but that the blood supply is still further impoverished by compression of the smaller arterioles in the bowel wall. Nutritional changes which may then occur vary from a superficial mucosal necrosis to gangrene of the whole thickness of the bowel wall with perforation and resultant peritonitis.

Simple occlusion, then, causes:

(*a*) Distension proximal and collapse distal to the site of occlusion with proximally a rise in intra-luminal tension.

(*b*) Increased peristalsis, then inco-ordinated and possibly reversed peristalsis, and finally inhibition of peristalsis.

(*c*) Increased secretion and decreased absorption of fluid above the site of occlusion.

(*d*) Increased gaseous content in the bowel above the occlusion, mainly from swallowed air, but also from bacterial fermentation and diffusion of gases from the blood stream.

(*e*) An increase in the bacterial content of the bowel above the occlusion accompanied by a change in the character of the flora.

(*f*) A variable degree of vascular damage to the bowel wall.

These are six constant pathological changes, varying only in degree. From them there follow many secondary effects, the nature of which depends upon the site of occlusion and the function of the bowel occluded.

High Small Bowel Occlusion. All the changes described above are present but the picture is dominated by the rapid severe loss of fluid and electrolytes from the circulation. It has been shown that even normally some 5–7 litres of fluid per day pour out into the upper reaches of the gastro-intestinal tract as digestive secretions, and to this must be added the fluid exudate produced by intestinal dilatation and the resultant circulatory congestion. If this mass of fluid is lost, either in the vomit or stagnating in the dilated coils of intestine, gross dehydration and chemical imbalance results.

The Secondary Effects of Dehydration

(1) Change in the Viscosity of the Blood

To compensate for the loss of fluid there is a withdrawal of fluid from the interstitial body tissues. This source of available fluid is soon exhausted, the volume of blood plasma begins to drop and there is an increase in the viscosity of the blood with a generalized slowing of the circulation. In cases of high obstruction there may occur a huge reduction in the oxygen content of the venous blood.

(2) Changes in the Chemistry of the Blood

Compensating in part for the fall in chloride, there is a sharp rise in the plasma bicarbonate. At the same time there is a rise in the blood urea and non-protein nitrogen. This is due to three things concentration on account of fluid loss, increased production and decreased elimination. Production is increased by increased tissue breakdown, either locally from vascular damage to the bowel wall, or generally as the result of anoxæmia and circulatory stasis accompanying dehydration Elimination is decreased by the reduction in the amount of urine excreted and by damage to the kidneys by circulatory stasis. Depletion of potassium is also important and can itself cause paralytic ileus It is less easy to demonstrate than depletion of chloride, for the potassium ion is mainly intracellular and the plasma level may remain unaltered.

(3) Changes in the Kidney

Although constant changes in the kidney have not been described, some interference with renal function is common, probably as a result of anoxic damage to the renal epithelium.

Low Small Bowel Occlusions

The function of the upper small intestine is mainly secretory, that of the lower small intestine mainly absorptive. The manifestations of low small intestinal occlusion are thus different in several aspects from those of high small intestinal occlusion. The onset of vomiting is later and the degree less marked. Instead, there is a growing abdominal distension as the small bowel fills up with liquid and gas. Today there is still room for discussion about the exact pathology and the lethal factors involved in low occlusions. It is now generally agreed that dehydration and chemical imbalance play an important part, even if there has been no vomiting at all, for the large volume of liquid lying unabsorbed in the distended small bowel is lost from the general circulation just as surely as vomited fluid. Some authorities, such as Robert Holt of Manchester (*1934*; 1939) have for many years stoutly maintained that this is the only factor of really vital importance. Others have been impressed by certain clinical differences between high and low occlusions and thought to explain them by assuming the presence of an additional *toxic* factor, claiming that in the evil-smelling, stagnant fluid in the occluded ileum a poisonous substance is produced, probably by the breakdown of protein, and absorbed into the general circulation with adverse effects. A mass of conflicting evidence about the nature, route of absorption, and action of this elusive toxin has been collected over the course of the last two or three decades and it does appear to the writer that it is not possible to exclude altogether the possibility that the combination of heightened bacterial activity in the intestinal lumen and vascular damage to the bowel wall may lead to the production and absorption of a chemical capable of causing circulatory depression. Nevertheless it is commonly thought today that the "toxæmic" manifestations of low small intestinal occlusion can all be explained on a basis of dehydration and electrolytic disturbance and that the apparent clinical differences between high and low occlusions are due to differences in the rate of fluid loss and the nature of the electrolytic, metabolic, and hormonal disturbance.

Occlusion of the Large Intestine

Occlusion of the large intestine is nearly always caused by a carcinoma, less often by diverticulitis. If the ileocæcal valve is competent and prevents reflux, the gaseous distension of the closed segment of large bowel leads to a rapid rise in intra-luminal tension and early embarrassment of the vessels in the bowel wall. If the ileo-cæcal valve is incompetent, there is a less rapid rise in tension in the large bowel but early onset of secondary ileal obstruction Lethal factors include dehydration and chemical imbalance associated with this ileal occlusion, perforation of the bowel due to patchy necrosis anywhere above the growth, but most commonly in the cæcum, and respiratory and circulatory embarrassment due to extreme abdominal distension.

Closed Loop Obstruction

Closed loop obstruction without strangulation is of rare clinical occurrence except for two conditions: obstruction of the large bowel in the presence of a competent ileo-cæcal sphincter and obstruction of the appendix. It can also occur in obstruction of a Meckel's diverticulum. Clinically the three phases of closed loop obstruction are most perfectly seen in a case of acute obstruction of the appendix The first phase may be ascribed to the stimulation of nerve endings and the reflex results of such stimulation.

Thus pain in the region of the umbilicus occurs, being central and not accurately localized, followed by nausea and vomiting. Later a phase of "toxæmia" occurs and clinically the patient may resemble a case of low ileal obstruction. The final phase is perforation and peritonitis, the severity of which depends upon the virulence and concentration of organisms within the appendix.

Intestinal Strangulation

Of the many varieties of acute intestinal obstruction, the most fatal are those in which the bowel is deprived of its blood supply. The term strangulation is often used to denote any case of this kind whether the blood supply is in fact truly "strangled" by an occluding band or the tight neck of a hernia or obstructed by thrombosis or embolism of the vessels in the mesentery. Strangulations are conveniently considered under three headings, according to the length of the loop strangulated. Other important factors are whether the loop is inside the general peritoneal cavity or outside it, for instance in a hernial sac, and whether the vascular occlusion is arterial, venous or both.

Long Loop Strangulation

Mesenteric Vascular Occlusion. This may be arterial, venous or both and thrombotic or embolic. Arterial occlusions are rather more common than venous. Although the original pathology may be thrombotic or embolic, primary thrombosis is rare, the pathology most often found being thrombosis on top of original embolism. The ætiological factors include endarteritis, atheroma and arteriosclerosis. Although one might expect that an ischæmic infarct would follow arterial blockage, this is rare, an hæmorrhagic infarct being by far the commoner result. Venous occlusions are nearly all thrombotic, some of the causes being local sepsis, cirrhosis of the liver and polycythæmia. In venous occlusions an hæmorrhagic infarct is also the usual result, with rapid capillary engorgement and the outpouring of a blood-stained fluid into the lumen and tissue spaces of the bowel and into the peritoneal cavity. Œdema of the bowel and mesentery soon becomes marked and if survival continues long enough can eventually lead to occlusion of the arterial circulation. As in arterial occlusions, the initial blockage is extremely liable to be followed by an extension of the thrombosis into the arcades and smaller veins.

The Cause of Death. In long loop strangulation death usually occurs before gangrene has been followed by peritonitis The cause is the sudden extreme loss of fluid which leads to early circulatory failure As a generalization one may say that the longer the loop the shorter the survival, though cases have been recorded of survival after occlusion even of the superior mesenteric artery

Medium Loop Strangulation

This term is usually taken to mean a loop 4–5 feet long Clinically, as in the long loop strangulation, the condition is not common and the most usual causes are mesenteric thrombosis or embolism or volvulus. Strangulation of a loop of medium length is attended by the same pathological changes as those described for a long loop, but as the withdrawal of fluid from the circulation is less rapid this phase may be survived and the patient live until gangrene and perforation of the bowel occur

Short Loop Strangulation

The commonest cause of short loop strangulation is strangulation in an external hernia. Other causes include constriction by bands and strangulation of an internal hernia. Pathology may conveniently be considered under two headings:

(1) Changes above the strangulated loop.
(2) Changes in the strangulated loop.

The changes occurring above the strangulated loop are precisely the same as those occurring in the case of simple occlusion at the same level. The changes in the strangulated loop are exactly the same as those already described as occurring in long and medium loop strangulation, the difference being merely one of degree. In the vast majority *the occlusion is venous, at least to start with, and the first phase is one of* increased capillary permeation with the outpouring of a blood-stained fluid into the lumen and tissue spaces of the bowel and into the hernial sac or peritoneal cavity in which the bowel happens to be lying. This loss of fluid is in itself unimportant but it must be remembered that the loss of fluid from above the loop may be of great importance particularly if the loop is a high one. Later, gangrene and perforation of the strangulated loop lead to spreading infection and it is possible that a circulatory depressant produced by septic autolysis of the bowel may exert some effect.

Functional Intestinal Obstructions

Functional intestinal obstruction may be paralytic or spastic or a mixture of the two Paralytic ileus may result from causes which are generalized or localized and may be classified thus:

(1) Generalized Causes.
  (*a*) Circulatory Failure
    (i) Pneumonia
    (ii) Uræmia.
    (iii) "Shock Syndrome." Dehydration Electrolytic imbalance. Potassium depletion.
    (iv) Other advanced toxic states
  (*b*) Neurogenic Causes. Splanchnic Irritation or a Reflex via the Splanchnic Nerves
    (i) Renal causes· trauma, renal colic, etc.
    (ii) Retro-peritoneal hæmatoma or cellulitis.
    (iii) Acute pancreatitis
    (iv) Any severe generalized trauma, particularly if painful, e g. Multiple fractures
  (*c*) Local Causes. (Damage to the neuromuscular structure of the bowel wall, causing paralysis through a direct local effect, as well as possibly through reflex inhibition of peristalsis via the splanchnic nerves and the general effects of circulatory depression and electrolytic imbalance):
    (i) Gross intestinal distension.
    (ii) Embarrassment *of the circulation in the bowel wall*

(iii) Trauma at operation; rough handling; cooling or drying of the exposed bowel.
(iv) Peritonitis.

Of these, local causes are the most common. Trauma at operation causes paralysis through a splanchnic reflex. Distension has the triple effect of initiating a similar reflex, of damaging contractile power of the intestinal muscle by the mere mechanical stretching and of causing local circulatory stasis with vascular damage. Peritonitis causes paralytic ileus on account of the circulatory depression of a bacterial toxæmia, by a reflex inhibition via the splanchnics as a response to peritoneal irritation, and directly by inflammatory changes in the bowel wall. Whatever the cause of a paralytic obstruction, in the absence of peristalsis the bowel becomes filled with fluid and gas and there is a steady rise in the intraluminal tension. The resultant stretching of the bowel wall reinforces other causes producing ileus and, just as in any other variety of acute obstruction, vascular changes soon appear, starting with capillary engorgement and œdema and ending with local necrosis of the bowel wall, perforation, and peritonitis. It will be seen that death in paralytic obstruction is intimately connected with the mechanical factor of distension. Even if the primary cause is a generalized one, this distension may well end in vascular damage and ultimately peritonitis. Thus, even if the cause of the paralysis cannot be treated, the treatment of the intestinal distension is clearly of paramount importance.

Spastic Functional Obstructions

Functional obstructions which are entirely spastic are rare, though a number of cases have been described. The causes include (Steindl, 1926):

(1) Injury to peritoneal nerve plexuses.
(2) Causes directly affecting the intestinal tract, trauma, etc.
(3) Irritation of intestinal contents.
(4) Hysteria
(5) A group of cases of unknown etiology

The pathology resembles that of simple occlusion, the occluding agent being a segment of bowel in spasm

Diagnosis

The three cardinal symptoms of acute intestinal obstruction are pain, vomiting, and intestinal distension, usually presenting in this order

Pain. In uncomplicated simple occlusions the cause of pain is hyperperistalsis in response to the rise of intra-luminal tension. The pain is therefore colicky, felt around or just above the umbilicus in small bowel obstructions and in the hypogastrium in large bowel obstructions. Strangulating obstructions give rise to waves of severe colic between which a steady pain is felt, occasionally referred to the back. Severe local pain also occurs in late cases complicated by peritonitis. With pain must be considered tenderness, guarding, and rigidity. Simple occlusions do not cause tenderness, unless gross stretching of the bowel is present, as in late occlusions of the large intestine. A strangulating

obstruction causes tenderness and muscle guarding, while the onset of peritonitis is indicated by tenderness, rebound tenderness, and reflex muscle rigidity.

Vomiting. Vomiting usually occurs reflexly soon after the onset of obstruction. Later, obstructive vomiting characteristically starts by consisting of clear fluid and undigested food, becoming bile-stained and finally dark brown and evil-smelling, the so-called "fæcal vomiting."

Abdominal Distension. Abdominal distension is maximal in low obstructions, including large bowel obstructions, and minimal in high obstructions. For anatomical reasons, the dilating small bowel tends to produce hypogastric distension, whilst a dilating large bowel gives rise to distension and hyper-resonance in the cæcal area and across the abdomen, at or above the level of the umbilicus.

Other clinical features include:

Absolute constipation, usually, though not invariably, present after perhaps one bowel action.

Changes in temperature, pulse and respiration are very variable and indicate complications of obstruction rather than obstruction iself, e.g. elevation of temperature with the onset of infection; elevation of pulse rate with a diminution of blood volume; embarrassment of respiration with gross distension and a raised diaphragm.

A dirty tongue and offensive breath appear early.

Visible peristalsis. Visible coils of bowel arranged in a "ladder pattern" may be seen if the abdominal wall is thin and atrophic.

A palpable mass, neoplastic or inflammatory, may be detected, or occasionally the outline of a single distended loop of bowel in a case of volvulus.

Auscultation of the abdomen may be important. The exaggerated, high-pitched tinkling sounds of an organic obstruction contrast with the absolute silence of a paralytic ileus.

Rectal examination and examination of the hernial orifices must never be omitted. It is, for instance, only too easy to overlook a strangulated femoral hernia in a fat woman.

Investigations

The diagnostic enema is of some value If obstruction is present, a result is often obtained with an initial enema but subsequent enemata are returned clear. While this method of investigation may be of use in some doubtful cases, clinical and radiological evidence is in most cases sufficient to make a certain diagnosis without it. Moreover, the value of subsequent X-rays is vitiated by giving an enema.

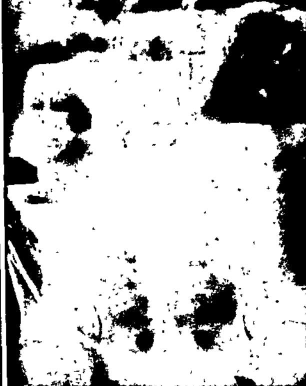
Leucocytosis does not mean obstruction but may occur if complications of obstruction are present, e.g peritonitis

Urinary Analysis gives an indication of the degree of dehydration and chloride depletion. Occasionally a paralytic obstruction is shown to be due to acute pancreatitis by a high urinary diastase

Radiological Diagnosis. Plain films of the abdomen in the supine and erect positions will show the distribution of gas and the presence or absence of fluid levels. Positive evidence on which reliance may be placed is available some six hours or so after the onset of obstruction and the site and type of obstruction can be deduced in many cases.



Supine



Erect

FIG 140 X-rays showing acute obstruction of the ileum, with typical distribution of gas in the supine film and multiple fluid levels in the erect film



Supine



Erect

FIG 141 X-rays showing obstruction of the pelvic colon, with the whole large bowel dilated with gas and, in the erect film, a long single fluid level in the cæcum Competent ileo-cæcal valve and no secondary ileal dilatation.



Supine



Erect

Fig. 142. X-rays showing large bowel obstruction with "blow-back" into the ileum through an incompetent ileo-caecal valve.



Supine



Erect

Fig. 143. X-rays showing small bowel obstruction. It is not uncommon in the supine film for gas to outline particularly one main dilated coil as in this case.



Supine

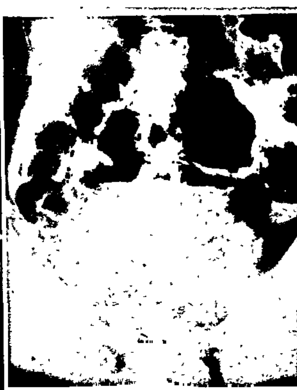


Erect

FIG 144. X-rays showing a single very large gas-filled loop and a single fluid level; volvulus of the pelvic colon.



Supine



Erect

FIG 145. X-rays showing volvulus of the cæcum which has rotated into the centre of the abdomen, distended with gas and with a long fluid level in it.

Treatment

Twenty years ago, the treatment of intestinal obstruction posed no awkward problems for once the diagnosis had been made, operation followed as soon as possible. Today the position is not quite so simple, for it has become abundantly clear that in some late cases of simple occlusion, dehydration and chemical imbalance can make the patient so ill that immediate operation carries a higher risk than delayed operation after preliminary treatment with intravenous fluids and gastro-intestinal suction. It is even true that in some cases occlusion, due primarily to adhesion, kinking and distension, may resolve spontaneously under such non-operative treatment. Three possible lines of treatment are thus available:

(1) Immediate operation.
(2) Delayed operation.
(3) No operation

Ideally, all strangulating obstructions are best treated by immediate operation, for the risk of allowing gangrene of the bowel to occur rules out any delay, all simple occlusions by delayed operation, after a variable period of intravenous fluids and gastro-intestinal suction, and all cases of uncomplicated paralytic ileus by suction and intravenous fluids, without operation. In practice, matters do not work out quite like this, for differential diagnosis is not sufficiently accurate to distinguish the various types of obstruction with certainty, particularly in the early stages

Thus all obstructions seen during the first 24 hours should be treated by early operation. If symptoms have been present for more than 24 hours, it is justifiable to delay operation in some cases. The only criteria for this delay are:

(*a*) No signs at all suggestive of a strangulating obstruction are present.

(*b*) Since strangulation cannot be rigidly occluded, the risk of delay resulting in gangrene of the bowel must be considered to be less than the risk of intervening while the patient is grossly affected by dehydration, electrolytic imbalance, and intestinal dilatation. Once suction and intravenous fluids have reduced the risk of operation, the relative merits of delay and no delay alter and the second criterion soon ceases to apply. In the opinion of the writer, it is often right to treat a late organic obstruction by a short period of suction and intravenous fluids, followed by operation, and occasionally obstruction is relieved by suction and operation proves unnecessary, but this is very different from setting out to treat obstruction with suction and with the avowed intention of avoiding operation altogether. This is a dangerous method and it must be recognized that valuable as the use of suction is, its misuse may be lethal.

Varieties of Gastro-Intestinal Suction. Gastro-intestinal suction of some kind plays a part in practically all intestinal obstructions. The tube employed may be a short tube or a long tube. The short tube, the tip of which lies in the stomach or upper duodenum will empty and keep empty the stomach and do a little to deflate the upper small intestine. Effective intestinal suction can only be achieved by using a long tube, such as the Miller-Abbott double lumen tube or the Harris single lumen tube. The former is passed into the stomach with the balloon collapsed or containing a little mercury, and once the pylorus is passed the balloon is inflated, suction is applied and the tip of the tube is carried down to the site of obstruction

The Choice of Tube. If immediate operation is decided upon, the stomach should be emptied by suction and a short (Ryle's) tube left *in situ*. It is of vital importance to keep the stomach empty during induction of anæsthesia, for the risk of aspiration of vomit into the air passages has been increased by the use of modern relaxants. If delayed operation is intended, again gastric or duodenal suction with a short tube is preferable to wasting time and exhausting the patient in the not easy technique of passing a Miller-Abbott tube through the pylorus. If paralytic ileus uncomplicated by an organic lesion requiring surgery is to be treated by suction and intravenous fluids, gastric or duodenal suction may well be insufficient and the long tube, passed under radiological control, may be essential.

How much Water and Salt? Half the battle in the treatment of intestinal obstructions lies in the elimination or prevention of dehydration and electrolytic imbalance. The whole question of fluid balance is fully discussed in Chapter IX and will not be further examined here, except to say that whether operation is to be immediate, delayed, or avoided altogether, early attention to this side of the problem is all-important.

Anæsthesia

Deep general anæsthesia is to be avoided. Local anæsthesia is suitable for some cases of strangulated hernia Laparotomy demands good muscular relaxation. Regional blocks and spinal anæsthesia were justly becoming popular in the late 1930s and early 1940s but today it seems probable that light narcosis and analgesia, supplemented by a relaxant provides the best form of anæsthesia.

General Surgical Technique

No attempt will be made to cover all aspects of surgery for intestinal obstruction, but certain important general observations may be made·

(1) Clinical and radiological evidence should indicate the site of obstruction sufficiently accurately to decide the most appropriate incision. This should usually be large, for no avoidable difficulties of access should be accepted

(2) Rough handling of the distended bowel, pulling on the mesentery, heavy retraction, and loss of warmth and fluid by evaporation from the surfaces of exposed viscera should be eliminated as far as possible.

(3) Evisceration is not in itself harmful. In the presence of gross distension it is often better to allow the dilated coils to prolapse out of the wound and be supported in warm, moist packs by an assistant, so that a clear view of the obstruction is obtained.

Post-operative Care

The general features of post-operative management are dealt with elsewhere. After an operation for intestinal obstruction the particular necessities are:

(1) Maintenance of fluid and electrolytic balance.

(2) Gastro-intestinal suction until normal peristalsis is re-established. It is of particular importance not to withdraw suction too early after resection of bowel for gangrene.

(3) The use of antibiotics to control or prevent peritonitis, which is a common complication.

If the bowels do not act for several days, no serious difficulties arise if dilatation of the small bowel is prevented by gastro-intestinal suction. The use of aperients and peristaltic stimulants, such as pituitrin, eserine and the like, is seldom effective and often harmful, while although an enema may be useful when tone has returned to the small bowel and the large bowel is distended with gas, it is merely exhausting to the patient if given at the wrong moment when abdominal distension is due to dilatation of the small intestine.

Individual Varieties of Acute Intestinal Obstruction

The following is a useful classification of intestinal obstructions (Rodney Smith, 1947).

(1) *Those due to development anomalies.*
- (*a*) Atresia and stenosis of the bowel.
- (*b*) Imperforate anus.
- (*c*) Compression by developmental bands.
- (*d*) Volvulus associated with mal-rotation of the mid-gut.
- (*e*) Obstruction caused by a Meckel's diverticulum.
- (*f*) "Meconium Ileus."

(2) *Other "Mechanical" Obstructions*
- (*a*) Strangulated hernia, external and internal.
- (*b*) Volvulus.
- (*c*) Intussusception.
- (*d*) Obstruction by bands or adhesions.
- (*e*) Intra-luminal impaction (e g. gallstone obstruction).
- (*f*) Pressure by a tumour outside the bowel.

(3) *Obstructions of Inflammatory Origin.*
- (*a*) Diverticulitis.
- (*b*) Ileo-cæcal granuloma
- (*c*) Post-inflammatory strictures

(4) *Obstruction by Intestinal Neoplasms.*
- (*a*) Primary carcinoma
- (*b*) Sarcoma.
- (*c*) Secondary deposits in the bowel wall
- (*d*) Benign neoplasms

(5) *Obstruction from paralysis of the bowel or interference with its blood supply.*
- (*a*) Paralytic ileus
  - (i) with peritonitis,
  - (ii) without peritonitis.
- (*b*) Mesenteric vascular occlusion

Certain of these demand individual consideration

Strangulated Hernia (External and Internal)

Strangulated external hernia is the most common cause of acute intestinal obstruction, the frequency of the various types being approximately: inguinal 55 per cent,

femoral 20 per cent, umbilical 15 per cent, ventral 5 per cent, rare hernias 5 per cent. The constriction producing strangulation may be the muscular or fascial boundaries of the hernial ring, the peritoneal neck of the sac, or, particularly in umbilical or ventral herniæ, a band or adhesion inside the sac. Volvulus of a loop of intestine inside a large hernia also occurs. Strangulation of part of the circumference of the bowel wall occurs particularly in a femoral hernia (Richter's hernia).

DIAGNOSIS

The earliest symptoms are usually referable to the hernia, which becomes tense, irreducible, painful, and tender, while abdominal colic, vomiting, and distension soon indicate obstruction above the strangulated loop. Later, the general effects of dehydration and electrolytic imbalance develop, and later still gangrene of the bowel wall gives rise to œdema and discoloration of the skin over the hernia and the onset of peritonitis.

Treatment. The strangulated bowel is directly approached through the hernial sac, which is usually entered at the fundus, the dark fluid exudate sucked or mopped out and the responsible constriction is sought and cut or stretched. The strangulated bowel can now be drawn into the wound to bring into view the bowel directly above and below the loop, and carefully examined to determine its viability which is assessed largely upon these factors.

	Viable	*Not Viable*
Colour	Red → dark purple	Black → green
State of the visceral peritoneum.	Peritoneal sheen still present, though there are, perhaps, small subserous hæmorrhages.	Lustreless, shaggy appearance of the bowel.
State of the mesentery	Œdema of the mesentery but pulsation felt in the larger vessels No thrombosis	Thrombosis of main vessels. No pulsation felt.
Reaction to warming	Colour improves from plum colour to red Peristaltic wave seen to pass along the loop	Colour fails to improve. Still no pulsation in the main vessels No peristaltic wave seen.

The two constriction grooves across the bowel where pressure has been maximal are particularly examined If the bowel is viable it is returned to the abdomen and the hernia repaired If not viable, resection and anastomosis for the small bowel or exteriorization of the rarely strangulated large bowel are the accepted methods of treatment A doubtful constriction groove on a viable loop can be invaginated by a ring of Lembert sutures.

Strangulated Internal Hernia

Strangulated internal hernia may occur:

(*a*) Through a defect in the diaphragm.

(*b*) Into one of the retro-peritoneal fossæ around the duodeno-jejunal flexure, ileo-cæcal region or pelvic meso-colon

(*c*) Through a defect, congenital or post-operative in a mesentery.

(*d*) Through the foramen of Winslow (aditus to the lesser sac);

(*e*) Into the broad ligament.

A complete pre-operative diagnosis is clearly unlikely, but a general diagnosis of acute intestinal obstruction without evidence on which to exclude strangulation should lead

to early laparotomy and release of the bowel, which is examined and dealt with in exactly the same way as in an external strangulation.

Obstruction by Bands and Adhesions. Post-operative Mechanical Obstruction

A congenital peritoneal band such as that often found associated with a Meckel's diverticulum may cause obstruction, but the most common and most important problem under this heading is post-operative mechanical intestinal obstruction. This can occur

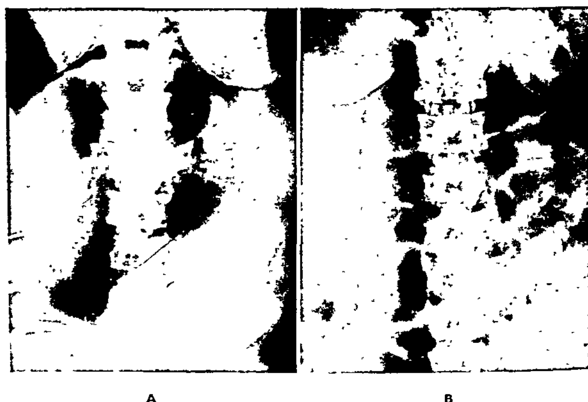


Fig. 146. (*a*) X-ray showing a single grossly dilated coil of jejunum necessitating reoperation ten days after laparotomy for a shell wound of the abdomen *Note* retained shell splinter and Miller-Abbott tube that would not enter the loop (*b*) Post-operative X-ray showing normal distribution of gas

in many ways, from adhesion, kinking and œdema, compression by inflammatory bands, œdema of an anastomosis, ensnaring of the bowel by mechanical factors concerned with the technique of the operation, volvulus brought about by adhesion of afferent and efferent limbs of an isolated loop The subject of post-operative obstruction is an immense one and there is room here for only certain important generalizations:

(1) Most post-operative obstructions are mechanical and not paralytic. To call them all "paralytic ileus" is one sure way of losing lives, though it is true that the resultant biochemical upset may cause interference with peristalsis and exaggerate the condition.

(2) Obstruction during the first few days after operation is usually due to non-strangulating causes, such as stomal œdema, or adhesive peritonitis with distension and kinking, and is often incomplete. Treatment with gastro-intestinal suction and careful control of dehydration and chemical balance will probably succeed, but radiological control is important and persistence of complete occlusion or the demonstration of a single coil

of distended bowel unaltered in consecutive films should lead to reopening the abdomen (Fig. 146).

(3) Mechanical obstruction occurring suddenly a week or more after operation is seldom relieved by any measure short of operation and is not infrequently caused by a band and strangulating in type, even though clinical features may not suggest this. It is thus advisable to waste no time but reopen the abdomen as soon as possible.

(4) Recurrent bouts of partial obstruction occurring intermittently after a widespread acute or chronic peritonitis call for gastro-intestinal suction, intravenous fluids and radiological control. Unless prompt relief of obstruction occurs, laparotomy is indicated for although strangulation is uncommon in this variety of obstruction it can occur..

Acute Obstruction by an Intestinal Neoplasm

Obstruction by an innocent neoplasm is rare, although intussusception in an adult may be caused by a submucous lipoma, for instance. Carcinoma of the large bowel is, however, a common cause of obstruction, particularly the malignant stricture in the pelvic colon or pelvi-rectal junction. Complete occlusion usually follows a period of increasing partial occlusion and is precipitated by impaction of fæces in the narrowed lumen and by œdema and inflammation. Gaseous distension of the proximal large bowel may become extreme and ultimately lead to patchy ulceration and even perforation, the cæcum being the most frequent site of this complication. The rapidity with which the rising intra-luminal tension is transmitted to the terminal ileum depends upon the competence of the ileo-cæcal valve.

Diagnosis. This does not usually present difficulties and plain X-rays are of very great value in determining the site of occlusion and excluding volvulus of the pelvic colon. In doubtful cases a barium enema under lower pressure will confirm the site, without risk (Figs. 147 and 148).

Treatment. Two opposite lines of treatment are available. Exploratory laparotomy may be performed, in the hope that a mobile carcinoma may be found and excision of the growth and relief of obstruction achieved by one operation, or failing this that some useful information may be obtained by palpating the growth, even if a preliminary colostomy is then preferred to immediate excision. Alternatively, laparotomy may be eschewed as unnecessary and even harmful, obstruction being relieved by a carefully sited colostomy, resection being performed on the unobstructed patient 2–3 weeks later. There are eminent supporters of each view and the following comments express the writer's own opinions:

(1) Immediate resection is occasionally indicated in the very early or partially obstructed case with minimal distension. In most cases resection is to be rejected, *not* because it presents great technical difficulties, but because distension of the bowel means shortening of the mesentery and it is very easy to accept a less than adequate lymphatic clearance. This applies to resection and end-to-end anastomosis and to the Paul-Mikulicz operation

(2) Palpation of the growth does not allow an accurate assessment of local operability, for a large and apparently fixed growth may be smaller and quite mobile once œdema and inflammation have subsided as a result of a proximal colostomy. Moreover the softened, œdematous bowel directly above the growth may quite easily be ruptured, with gross contamination of the peritoneal cavity and probable death of the patient.



A



B

FIG 147 Plain X-ray and barium enema showing obstruction of the descending colon by a neoplasm



A



B

FIG 148 (*a*) Plain X-ray from a patient with acute obstruction two years after hemicolectomy for carcinoma of the cæcum The short gas-filled segment of transverse colon suggests a new primary carcinoma of the splenic flexure (*b*) Barium enema confirms this

(3) There is thus a very good case for treating all but the earliest cases by a proximal defunctioning colostomy, not cæcostomy, without exploration, planned resection being carried out 2–3 weeks later.

(4) Carcinoma of the cæcum, ascending colon, or hepatic flexure is less likely to cause complete obstruction than carcinoma of the distal large bowel. When it does, one stage hemicolectomy can usually be performed with safety and adequate lymphatic removal. Occasionally ileo-transverse colostomy may be preferred as a first stage, paving the way for later resection.

Mesenteric Vascular Occlusions

Occlusion of the mesenteric blood vessels supplying part of the intestine is a catastrophe which causes one of the most fatal of all varieties of intestinal strangulation. The occlusion may be embolic or thrombotic and the causative factors include:

(1) Embolic:

(*a*) Endocarditis.

(*b*) Atheroma of the aorta.

(*c*) Arteriosclerosis.

(*d*) Any disease in which thrombi form and may become detached into the general circulation.

(2) Thrombotic:

(*a*) Any stenosis of the portal system, splenic anæmia, cirrhosis of the liver.

(*b*) After certain operations, especially acute appendicectomy, splenectomy, pelvic operations.

(*c*) After trauma to the mesentery, especially after volvulus, intussusception, or strangulation in a hernia.

(*d*) Rarely as a result of a mesenteric neoplasm, e.g a lymphoblastoma.

Pathology. Mesenteric occlusions may be classified as:

(1) Arterial, accounting for 51·2 per cent of cases in the series of Bowen and Felger (1942), which may be subdivided into:

(*a*) Embolic.

(*b*) Thrombotic.

(2) Venous Thrombosis, accounting for 42·8 per cent of cases in the same series.

(3) Arterial and Venous Occlusion, accounting for 6 per cent of cases in the same series.

Arterial embolism is commoner than spontaneous thrombosis but after embolism of a main vessel has occurred extension of the arterial occlusion proceeds by spreading thrombosis into the smaller vessels. Primary arterial thrombosis due to atheroma or endarteritis of the mesenteric vessels themselves is rare. Arterial or venous occlusion is followed by hæmorrhagic infarction with œdema and distension. Spontaneous recovery of the bowel is possible if occlusion of a main stem is not followed by rapid thrombotic extension into the small arcades thus destroying all chance of a collateral circulation and making gangrene certain.

Diagnosis. Severe colicky pain and vomiting are accompanied by extreme peripheral circulatory failure due to the loss of fluid from the circulation into the lumen and tissue spaces of the infarcted bowel and peritoneal cavity. Abdominal rigidity, tenderness, and rebound tenderness are present and ascites is often detectable clinically. A palpable tumour is occasionally found and a high leucocytosis is usual.

Treatment. Laparotomy should be supported by energetic treatment of shock, which may be extreme. Blood transfusion is nearly always called for.

Intestine which is gangrenous must clearly be resected, but the treatment of engorged and œdematous intestine short of gangrene depends upon the site and extent. A limited infarct of the small intestine should be treated by resection and anastomosis, of the large intestine by exteriorization. The routine use of anti-coagulants has had a profound effect upon prognosis in these cases, in preventing extension of thrombosis into neighbouring mesenteric vessels. Very extensive infarction has in the past led to resection of even 18 or 19 feet of small intestine, but only very occasionally has recovery followed. It is now believed that in the absence of actual gangrene the best hope of success lies in avoiding resection in these very extensive infarctions and treating the patient with general measures and anti-coagulants.

Volvulus

Acute volvulus of the small intestine is rare, though strangulation of a loop of bowel may sometimes be complicated by rotation of the loop. Laparotomy allows it to be released and untwisted. Viability is then assessed and if the bowel is considered not viable resection and anastomosis is carried out.

Acute volvulus of the large bowel most commonly involves a long redundant loop of pelvic colon. Rapid extreme distension is a feature of this form of obstruction and plain X-rays confirm the diagnosis (Fig. 144). At operation the bowel is untwisted and a rectal tube passed by an assistant guided into the dilated loop. A non-viable loop is exteriorized

Intussusception

The telescoping of a segment of bowel into that distal to it, intussusception, may occur with or without a demonstrable cause. Infrequently, a simple tumour, such as a submucous lipoma projects into the lumen and forms the apex of an intussusceptum. This may occur at any age and in large or small bowel.

Idiopathic Intussusception, with no demonstrable cause, occurs mainly in babies or young children, the apex of the intussusceptum (the invaginated bowel) being small intestine or the ileo-cæcal valve, though this may pass through the intussuscipiens (receiving bowel) even as far as the rectum If untreated, the blood supply of the intussusceptum is soon cut off by kinking and compression of its mesentery and œdema and inflammation, producing irreducibility and perforation Whilst it is in theory possible for the whole intussusceptum to slough away and be passed per rectum, with spontaneous cure, this is in the highest degree unlikely and the usual result of an untreated intussusception is peritonitis and death

Diagnosis. The patient is often a baby boy, 6 months or so in age. Attacks of abdominal colic during which the baby turns pale and screams, drawing up his knees, are separated by periods during which he appears normal An early vomit is usual and

as time progresses he begins to show signs of exhaustion and looks ill. Feeds are usually refused. One normal stool may be passed, but later blood and mucus per rectum indicate the extending vascular engorgement of the intussusceptum. It is important to realize that this sign may not be present for 12 hours after the first symptom. The story should suggest intussusception at once and examination of the abdomen must be careful and unhurried. The diagnosis is made by the discovery of a mass which often becomes suddenly much more clearly defined as the muscle of the bowel wall goes into spasm ("hardening under the hand") and the surgeon should not wait for the later sign of blood on the finger stall on rectal examination. Dance's sign, emptiness of the right iliac fossa, is inconstant. Radiological examination may help in a doubtful case.

Treatment. Laparotomy and reduction of the intussusception by "milking," squeezing the intussusceptum back by pressure on the intussuscipiens just beyond the apex, is usually considered the most suitable treatment. The irreducible intussusception must be resected, and end-to-end anastomosis is then preferable to enterostomy, which a baby tolerates badly.

Reduction with an Opaque Enema

It is perfectly possible to reduce many intussusceptions without operation by the pressure of an opaque enema watched on the fluorescent screen. In skilled hands this is a justifiable mode of treatment, but many hold that operation is to be preferred because:

(*a*) Clearly only reducible intussusceptions can be treated with the opaque enema. This class of case presents no problem, for surgical relief is simple, rapid, and the certainty of complete reduction at once visible.

(*b*) It is less easy to be certain of the *completion* of reduction by the opaque enema, particularly if the intussusception started in the small bowel above the ileo-cæcal valve.

Gall Stone Obstruction

Gall stones large enough to obstruct the intestine have nearly always entered the gastro-intestinal tract through a fistulous communication, ulcerating through from the fundus of the gall bladder usually into the duodenum. As the stone passes down the intestine it often becomes recurrently held up and then released and characteristically the story is, therefore, one of intermittent bouts of colic and vomiting, culminating in complete obstruction when the stone reaches the ileo-cæcal valve The history thus often suggests the diagnosis, while plain X-rays, as well as demonstrating the existence of obstruction, may also show the stone itself or gas shadows in the biliary tree.

At operation the impacted stone cannot usually be broken up and enterotomy and removal normally prove necessary.

Functional Obstructions

Apart from general peritonitis, discussed fully elsewhere, paralytic ileus is a rarity, though it may occur as a complication of various conditions associated with shock, or other extreme circulatory derangement It may thus follow any severe injury, particularly with multiple fractures, and complicate acute pancreatitis, retroperitoneal hæmatoma, renal injuries, pneumonia, uræmia or the crush syndrome. The treatment starts

naturally with the treatment of the cause and in addition the bowel must be prote from uncontrolled dilatation by gastro-intestinal suction and the circulation fi *dehydration or chemical imbalance by intra-venous fluids.*

References

Rodney Smith (1947) *Acute Intestinal Obstruction*, Edward Arnold & Co., London.
Holt, R. L. (1934) *Lancet*, 1, 724.
Idem. (1939) *Ibid*, 2, 61.
Steindl, H. (1926) *Arch. f. Klin. Chin.* 139, 245.
Bowen, A. and Felger, L. (1942) Mississippi Valley M. J. 64, 24.

CHAPTER VIII

THE APPENDIX

J. H. Lees Ferguson

Historical Note

The appendix, as an anatomical structure, was recognized by Pharaonic embalmers. It was illustrated by Leonardo da Vinci, described by Fallopius and Vesalius, and to Verheyen is credited its name. An organ of insignificant size and of negligible function, it remained in the penumbra of mediæval anatomy and surgery until the eighteenth century, when the acute inflammation which attacks it was accurately recorded by Heister in 1755 and by Mestivier in 1759. Credit for the first successful appendicectomy belongs to Claudius Amyand of St. George's Hospital. The operation was performed in December, 1735, and reported in the Philosophical Transactions of the Royal Society, 1736, under the title:

> "Of an Inguinal Rupture with a Pin in the Appendix Cœci incrufted with Stone; and fome Obfervations on wounds in the Guts."

Acute appendicitis was not, however, diagnosed with frequency till the latter twenty years of the nineteenth century, following a paper by Fitz of Boston, when Lister's revolution had already made an attack on the organ less hazardous. Shepherd (1954) has recently published an excellent historical review.

Development. At birth the appendix is found at the apex of the cæcum. Asymmetrical growth of the lateral cæcal wall and further descent places the appendix medially during the ensuing months. Rotation of the gut takes place during the period in which [illegible] occur, the appendix will remain [illegible] will be found under the spleen, the stomach or the liver, with the cæcum lying transversely, or at any subsequent point on the path of the cæcal descent. Should the descent be excessive, the appendix will lie in the right pelvis, and if the cæcal mesentery is unusally long, it will lie across to the left and may sometimes be found in the sac of inguinal or femoral herniæ. During the descent and enlargement of the cæcum, the appendix has the opportunity to achieve one of several positions, the incidence of these positions has been assessed as:

(1) Retrocæcal, 64 per cent.
(2) Pelvic, 32 per cent.
(3) Subcæcal, 2 per cent
(4) Pre-ileal, 0 9 per cent.

Two small operation series recently examined by the author showed more appendices in the sub-cæcal, para-cæcal, pre-and post-ileal positions than in the true pelvis.

Other abnormalities of Development. Absence of the appendix has been noted. Reduplication of the lumen of the whole organ is recorded and double appendices arising from differing origins have been seen. One of them is often associated with a tænia of the cæcum Congenital and acquired diverticula are discussed by Wilson

(1950). For a vestigial structure the appendix has nevertheless a remarkably constant anatomy.

Degeneration. It is important to realize that the appendix undergoes a physiological degeneration in middle life, irrespective of intercurrent inflammation. By this process, the organ is reduced in size, lymphoid tissue diminishes, but the lumen remain patent. The obliterated fibrous cord found in middle life in a patient who is not aware of symptoms, is the result of chronic inflammation.

Anatomy The anatomy of the appendix is too well known to require exposition. Accessory blood supply by a branch of the posterior cæcal artery may on occasion be of surgical importance.

ACUTE APPENDICITIS

Incidence. The incidence of acute appendicitis in this country is variously assessed as between one in 700 and one in 200 per annum. Males are more commonly affected than females. The disease has been recorded in new born infants and in centenarians, but is more frequent in the young than in the old (peak of incidence 12–30); it affects the eaters of meat more than vegetarians, the sedentary workers more than the active.

Mortality. The deaths from acute appendicitis showed a constant decline in England and Wales from a total of 2,722 in 1939 to 1,246 in 1949. This has been attributed to the introduction of antibiotics: but the better education of the public and their more willing entry into hospital, the greater accessibility of surgery, the earlier diagnosis by their doctors, and improved management of appendicitis in hospital must also be considered. On the other hand, there has been no improvement recorded in the deaths of children under the age of five in this country. In 1930 this was 130 out of a total of 3,000 deaths, and in 1950, 142 out of a total of 1,300. This is more attributable to difficulty in diagnosis than to failure of hospital treatment.

Ætiology

DIET. The influence of a high protein diet with a low residue is universally acknowledged as a cause of appendicitis. Examples are quoted in the many treatises on this subject of the abrupt rise in incidence when a population accustomed to vegetarian fare changes its dietary habits. The experiments of Wilkie on the occlusion of the artificial appendix in the cat give an indication of the increased virulence of bacterial infection associated with a meat as opposed to a carbohydrate diet. Stasis of cæcal contents has been adduced as a contributory factor in the development of acute appendicitis which may be seen in conjunction with organic obstruction of the colon. Constipation, poor or incoordinated cæcal function may also produce retrograde passage of fæces into the appendix, or prevent emptying.

ABNORMALITIES OF THE APPENDIX

Lymphoid Follicles. The natural history of the lymph follicles in and below the mucous membrane is significant in considering the ætiology of acute inflammation The appendix is endowed with its full number of follicles at birth Lymphoid activity is at its maximum in adolescence and decreases after the age of thirty when the incidence of appendicitis is still high. By this age, however, the mechanical or obstructive factors, in particular stricture due to previous inflammation, are more likely to be present. There

is no doubt that lymphoid tissue is a portal of entry for infection, and the swelling produced by lymphoid hyperplasia will produce obstruction in the appendix of a child. It has, however, a minor place in causing appendicitis in middle-age.

Variations of Structure. Fibrous stenosis, or stricture of the appendix, may delay emptying mechanically, or by interference with the normal rhythmic peristaltic movement, such delay being occasionally due to congenital abnormal folds. The reduction that takes place in the number of mucous glands in later years may further diminish mucus secretion and cause inspissation of fæces.

Coproliths or Fæcoliths and Foreign Bodies. Although the true calcified fæcolith is rarely found in cases of acute appendicitis, hard pellets of fæces which the organ has been unable to expel are frequently seen. There may be some obvious structural reason for their retention, but this is by no means always the case.

TRAUMA. Acute appendicitis has followed abdominal trauma in many recorded instances. Though this may be coincidental, the diagnostic possibility must be considered.

Pathology. The usual route of infection is through a breach in the mucosa, so that in the first instance the inflammatory process is localized. Whether infection occurs through the abrasions which undoubtedly occur from time to time depends on the virulence of the organisms present in the bowel and the patient's powers of resistance. Blood stream infection of the appendix has often been postulated, but is not generally regarded as of importance. The initial response to infection consists of vascular dilatation, followed by the emergence of the inflammatory exudate which swells the tissues and may fill the lumen. At the same time mucus production by the epithelial lining is stimulated, and the term *catarrhal appendicitis* is sometimes applied to the organ uniformly inflamed which is filled with muco-pus. A catarrhal inflammation is, however, confined to the mucous membrane and the infection usually extends beyond this at an early stage. If the lumen of the appendix remains patent resolution may take place with discharge of the infected contents into the cæcum, though more often there is a certain amount of organization of the exudate leading to fibrous thickening of the appendix wall If the infection is virulent the muscular and serous coats are involved and local peritonitis will occur, possibly with the formation of small abscesses in the wall in addition. It is even possible for a very acute infection to produce gangrenous changes without any evidence of obstruction being present Inflammation, when obstruction to the appendicular lumen exists from fibrous strictures due to previous catarrhal inflammation, gross lymphoid swelling, angulation, stenosis, or fæcal concretions, will result in *obstructive appendicitis* ("closed loop obstruction"). Swelling initially due to œdema is accentuated by obstruction to the venous drainage of the appendix wall. Further œdema and intraluminal exudation will then obliterate the arterial supply if this has not already occurred from infective thrombosis in the mesentery. The serosa is permeable to organisms early, owing to anoxia, and peritoneal irritation and infection is rapid. The usual response of the peritoneum consists in secretion of exudate at first clear, then becoming turbid with leucocytes Adhesive fibrin is deposited on the neighbouring structures, matting them together, and anchoring the omentum to the area. In children, the aged, and those debilitated by prison camp or intercurrent disease, response is poor, and general peritonitis the sequel. Though the short omentum is quoted as the anatomical reason for failure of localization in children, the main factor is undoubtedly a poorly developed inflammatory response. Should peritoneal reaction be well developed

before the distended appendix becomes gangrenous and perforated, an appendix mass is formed. Rupture of the appendix results in further pus formation and produces an appendix abscess, which may resolve, burst, or require drainage. When the onset of an inflammatory obstruction is rapid, and assisted by a purgative, or the peritoneum fails in its function, perforation of the appendix produces a localized peritonitis in the right iliac fossa, which becomes general in a very short time. The relief of symptoms and the temporary diminution in signs which accompany this event constitute a classical pons asinorum in diagnosis. For the pathological course which the peritonitis may pursue, the reader is referred to Chapter IX.*

Acute Appendicitis—Clinical Features and Diagnosis

The presentation of acute appendicitis described by J. B. Murphy as a sequence of events comprising:

(1) Generalized abdominal pain,
(2) Nausea and vomiting,
(3) Localized pain and rigidity in the right iliac fossa,
(4) Fever and leucocytosis,

remains a 40 year old touchstone in diagnosis. At the same time, the protean disguises in which appendicitis may present make any thinking surgeon distrustful of aphorisms in the diagnosis of this condition.

Symptoms. The exemplary history of acute appendicitis is that of a young man whose bowel has not been opened in the morning and who later suffers the onset of a diffuse abdominal pain, vaguely localized to the umbilicus. This pain, colicky in nature, is due to distension of the appendix by lymphoid swelling or mucoid secretion, and is referred segmentally to the tenth thoracic nerve. Nausea follows, presumably a sympathetic nervous reflex, and vomiting may supervene at this stage. The shift of maximal pain from the centre of the abdomen to the right iliac fossa may be noted by the patient, or elicited on questioning. Its intensity and precise site are variable, depending on the individual patient, the stage of inflammation and the position of the appendix. This pain is due to parietal peritoneal irritation, is constant, and when established, well localized. Though resolution of symptoms may occur at this juncture by discharge of the appendicular contents into the cæcum, obstruction of the appendix and impairment of its blood supply will more commonly cause an increase of pain until perforation occurs. A remission of pain is then noted, but the patient continues to feel ill, the pulse and temperature rise, and a rigor may be experienced. The temporary relief of pain is then succeeded by the diffuse burning discomfort of peritonitis, accompanied by resumption or increase of vomiting.

Signs. The patient suffering from acute appendicitis does not at first look ill. He may assume a low position in the bed, sometimes finding comfort in lying on the affected side. The pulse rate and temperature are by no means invariably raised in the initial stages, but may ascend as the peritoneum becomes involved. The tongue is furred and the breath carries a characteristic sweet fæcal factor. The abdomen is seen to move with respiration more freely on the left than on the right side. The contour will, however,

* The help of Dr. A. C. Thackray of the Bland Sutton Institute of Pathology in collaborating in the above section is gratefully acknowledged.

be normal unless peritonitis has caused distension, or a thin abdomen allows an inflammatory mass to be seen. Light palpation will show an increased tone in the muscles of the right lower quadrant. If peritoneal irritation is present from an appendix lying superficially, rigidity and rebound tenderness may be elicited. Deep palpation demonstrates the point of maximum tenderness close to McBurney's point. In the late stages of appendicitis a mass may be felt, diffuse and doughy, or well demarcated. Rectal examination gives rise to rectal and to abdominal pain, in the majority of cases, when the finger is pressed against the highest point on the right wall of the rectum which can be reached. A pelvic appendix, or later, an inflammatory mass may be felt. Percussion seldom gives positive information in early acute appendicitis. The cæcum is sometimes found to contain more gas than normal, but if cæcal distension is marked, a diagnosis of large bowel obstruction must be considered. Auscultation in the early phase of inflammation may demonstrate a minor hyperperistalsis due to local irritation. This is succeeded by disappearance of the borborygmi of onward peristalsis, and finally by cessation of the residual tinkling sounds. Hyperæsthesia of Sherren's triangle is found during the period of appendicular distension.

Classical Signs. A remarkable aggregation of eponymous and specialized signs have been associated with acute appendicitis. Their value, except in the confirmation of a diagnosis already strongly suspected, is debatable, though some are of use in attempting to forecast the position of the appendix, and they have been omitted in this chapter.

It is far more pertinent in the diagnosis of appendicitis to review the history critically, to think with one's fingers while conducting the examination, and to direct one's energies to the exclusion of some of the more common differential pathological states than to rely on the elicitation of specific tests which can only confirm inflammation in the right iliac fossa.

Variations of Symptoms and Signs. It is proper to reiterate the infinite diversity of presentation engendered by acute appendicitis. The factors producing these variations are the age, condition, and resistance of the patient, the virulence of the infection, the degree of appendicular obstruction, the position of the appendix and the taking of purgatives It falls to the lot of every surgeon during his career to remove a gangrenous pelvic appendix from a patient whose temperature and pulse are normal, and whose belly is soft Conversely, he may sometimes be surprised by the pale pink organ which he exposes following a history which is typical and signs which are of some severity.

Appendicitis in Childhood. The constitutional disturbance is more profound in children than in adults, the temperature often rising to a high level and the pulse rate being greatly increased. Vomiting or diarrhœa may be the presenting symptoms, preceding the pain. The abdominal signs are less obvious and less easy to elicit. The presence of respiratory infection in the form of streptococcal tonsillitis, pneumonitis, acute bronchitis or pneumonia may confuse the examiner, but should not deter him from making a diagnosis of appendicitis if definite lower abdominal signs are present. Co-existence of chest and appendicular inflammation should be recognized as a pathological possibility. An appendix mass is rarely seen in childhood, early perforation and peritonitis being more common than in adults.

Appendicitis in the Aged. The liability to perforation of the elderly patient's appendix, due to previous fibrosis or stricture formation, and to poor peritoneal reaction,

is well established. The majority of these patients present either with an established lower abdominal peritonitis, or with an appendix mass (presentation with a mass was a feature of McPherson and Kinmonth's older patients, but in two small recent series examined by the author, it was infrequent).

The history of onset of the attack is commonly short, and the pain seldom evokes the same degree of complaint as in younger patients. Pulse and temperature are often normal, fever and tachycardia being related to the later stage of peritonitis. Inability to pass flatus and abdominal distension are occasional symptoms which may misdirect the examiner's thoughts toward obstruction. Not infrequently the clinical picture is that of a silent small gut obstruction, secondary to pelvic peritonitis. The deceptive mildness of abdominal signs, absent as soon as perforation has taken place, render the evaluation of these patients even more difficult. There can be few surgeons who have not to their credit an older patient who has felt so much better following perforation that he has had to be persuaded into hospital unwillingly; in whom the only positive sign has been that of pelvic tenderness, and in whom operation has shown a perforated appendix with pelvic peritonitis

Obstructive and Catarrhal Appendicitis. The distinction between these two varieties of the disease is only possible in exemplary cases. In catarrhal appendicitis the onset is gradual. The central abdominal pain is vague and often without the periodicity of colic. While nausea is usually present, this may not progress to vomiting. The signs in the right iliac fossa are slow to develop. The patient with an obstructed appendix will generally remember a previous attack His central and upper abdominal colic is classically severe and vomiting an early and salient feature in the history. Pain in the right iliac fossa is progressive in severity, and finally incapacitating, until perforation occurs. During the onset of the condition pulse and temperature continue to rise and the local signs increase in extent and intensity.

Position of the Appendix. In peri-ileal and sub-cæcal appendicitis the localized pain due to peritoneal irritation is indicated by the patient's index finger at a position approximating to McBurney's point Maximal tenderness by one finger palpation will also be found here. The retro-cæcal appendix will give rise to pain well lateral to this, in the flank or loin, and such tenderness as is found may be most acute at a point just medial to the anterior superior iliac spine, or even in the renal angle, while rigidity is rare. The undescended appendix may manifest its signs and symptoms in the region of the gall bladder

The pelvic appendix is notorious for its diminished symptoms and absent abdominal signs. Pain is usually hypogastric and the other symptoms in this type of appendicitis are often caused by irritation of neighbouring structures in the pelvis Ureteric colic and microscopic hæmaturia, urinary frequency from inflammation of the vesical peritoneum, and diarrhœa or rectal pain from proximity to the sigmoid colon or rectum are all presenting features on occasion

Purgatives The taking of a purgative during the onset of acute appendicitis accelerates the tempo of the attack The central abdominal colic becomes more violent, the vomiting more severe, and the pain in the right iliac fossa is intensified. Rapid perforation is the sequel.

Antibiotics. Premature use of antibiotics may so modify the clinical picture as to cause a mis-diagnosis

Accessory Aids to Diagnosis. Accessory investigations are of more help in excluding other conditions than in establishing the diagnosis of acute appendicitis.

Radiology. Radiology in emergency may be of positive assistance. A plain abdominal X-ray will sometimes show increased gaseous collection in the terminal ileum and adjacent intraperitoneal fluid. (Thomas and Williams, of Cardiff Royal Infirmary, have given a striking demonstration of this at the British Medical Congress (1952). Help may also be gained in locating the cæcum and appendix, if this is abnormally placed.

The leucocyte count, if raised, signifies that the patient is suffering from acute inflammation, not necessarily of the appendix. A normal count is of no significance in excluding acute appendicitis, since a rise may not be demonstrable till the disease has been present for 24 hours or more, and in patients with poor resistance a rise may never occur.

Serial white cell counts are useful in gauging the progress of an appendix mass. Lowered values will be obtained if antibiotics are being employed.

Urine. The urine should always be examined by the naked eye, the nose and the microscope, in every patient suffering from acute abdominal symptoms.

Differential Diagnosis of Acute Appendicitis

A formidable list may be compiled of conditions which can simulate or are simulated by acute appendicitis. Unless the presence, alone, of one of these diseases can be established firmly and with certainty, the patient must be regarded as suffering from acute appendicitis, and treated accordingly. The traditional trio of disorders causing confusion most often is that of acute pyelitis, acute gastro-enteritis, and acute salpingitis. To these should be added the group of mesenteric glandular inflammations and, fifth, other acute conditions of the stomach, gall bladder, and large bowel

Acute Inflammatory Diseases of Ileo-cæcal Region

Acute Gastro-enteritis. The colic of acute enteritis classically precedes the vomiting, this order being reversed in appendicitis. Little reliance can be placed on the order of precedence of these symptoms. The pain of enteritis may localize to the right iliac fossa when the cæcum becomes inflammed or distended Diarrhœa, a cardinal symptom of enteritis, may also be caused by an acutely inflamed appendix. If local signs suspicious of appendicitis are found, this diagnosis should be made, bearing in mind the not infrequent co-existence of the two diseases

Acute mesenteric adenitis may be secondary to enteritis, or part of a generalized acute lymphadenopathy initiated by streptococcal infection or infective mononucleosis. Abdominal or inguinal glands may be felt, but the abdominal signs are not of a sufficient degree to account for the high temperature, rapid pulse, headache, photophobia or other disturbances In the early stages of mesenteric glandular inflammation, the leucocyte count will not be of help and, again, if there are local signs suggestive of appendicitis, operation is the safest course

Acute regional ileitis—Crohn's disease Acute Crohn's disease may simulate appendicitis so nearly as to be more frequently found at laparotomy than diagnosed pre-operatively. Ileal thickening may occasionally be felt on examination of the patient in bed, or under the anæsthetic. Removal of the appendix when regional ileitis is present almost inevitably gives rise to a fæcal fistula.

Ileo-cæcal tuberculosis most commonly presents with a mass. Inflammatory symptoms

and signs may predominate, particularly if the glands in the area are secondarily infected and necrotic. Radiography of the chest, and a plain X-ray of the abdomen (showing calcified glands) may be helpful in establishing the diagnosis.

Typhoid fever is now a rare disease in Britain. Paratyphoid fever, not uncommon in institutions, has been seen to simulate appendicitis in every respect, the patient being preserved from laparotomy by her previous appendix scar. Leucopenia is usually found by the time abdominal symptoms are manifest.

Amœbiasis. Acute cæcal amœbiasis is an important differential diagnosis in areas in which the disease is endemic. Operation on such cases without anti-amœbic treatment seldom fails to produce a fæcal fistula. Pre-operative examination of the stools is therefore essential. A smear should be taken from all appendices removed in the Tropics.

Acute phlegmonous cæcitis is classically indistinguishable from acute appendicitis, and has recently been well reviewed by Tagart.

Meckel's diverticulitis is a rare condition seen in children. A pre-operative diagnosis may be made by the mid-line hypogastric pain, a palpable fusiform mass below the umbilicus, or by increased pain on moving the navel upwards.

Diverticulitis of the cæcum. Inflammation in larger solitary diverticula of the cæcum causes a picture indistinguishable from appendicitis.

Carcinoma of the cæcum and ascending colon. The part played by organic colonic obstruction in producing a secondary appendicitis is well recognized. It is also not unusual for a growth of the cæcum to present as an inflammatory mass in the right iliac fossa, or by perforation and peritonitis. These facts must be considered in the diagnosis of appendicitis in the elderly. The history, the consistency of the mass, the finding of altered blood on sigmoidoscopy, and the detection of a microcytic anæmia are of help in establishing the diagnosis.

MECHANICAL AND VASCULAR AFFECTIONS OF THE ILEO-CÆCAL REGION

Volvulus. A complete volvulus of cæcum and terminal ileum is usually recognizable by its clinical and X-ray characteristics A partial volvulus of the lower pole of the cæcum may, however, resemble sub-acute appendicitis; the pain is a left-sided colic.

Intussusception. Ileo-cæcal intussusception may be erroneously diagnosed as acute appendicitis when there is no palpable mass, or blood in the stool. A useful analysis of presenting symptoms has been made by Elliott-Smith and Ward-McQuaid.

Internal hernia. This is distinguishable in its early stages by the severe and obstructive picture which it presents, combined with lack of abdominal rigidity. In its latter phases, with established peritonitis and a mass, the diagnosis may be difficult unless the history of onset is considered in detail.

Mesenteric embolism and thrombosis The less typical forms of embolism and thrombosis in the vessels of the terminal ileum, in particular those due to atherosclerosis, may be distinguished by the continual severe central pain and the degree of shock to which they give rise. When gangrene of the bowel and peritonitis have supervened, small gut distension and obstruction are usually recognizable on clinical and radiological examination.

Omental torsion. Torsion of the omentum may occur spontaneously, or in association with a hernia. The pain is central and severe, the signs not initially remarkable. The tender mass may sometimes be felt.

Torsion of an appendix epiploica—a rare occurrence in stout patients—cannot be diagnosed with certainty by external examination.

OTHER ACUTE INTRA-ABDOMINAL INFLAMMATIONS

Perforated Peptic Ulcer. Leakage of gastric contents from a perforation of the duodenum down to the right iliac fossa may sometimes give rise to a difficult diagnosis, particularly when a history of previous dyspepsia cannot be obtained. The relation of the onset of the attack to a meal may be significant. Prostration, shock, and local signs are more pronounced and of rapid onset, appendicitis very rarely gives rise to tenderness at the level of the trans-pyloric plane. Sub-diaphragmatic gas is far more commonly due to perforation of an ulcer.

Acute cholecystitis and torsion of the gall bladder. These conditions cause no difficulty when the gall bladder is in its correct position, and subcostal tenderness is found. An acutely inflamed gall bladder which is hanging on a long pedicle in the right iliac fossa and there gives rise to peritonitis, may not be diagnosed until it presents in the wound. As in appendicitis, neither the very old nor the very young are immune from acute cholecystitis.

Acute pancreatitis seldom gives rise to right-sided pain and rigidity. Shock, cyanosis, and pain in the back are constant features. The serum amylase test should be performed within 24 hours of the onset of the attack and values below 500 units should be accepted with reserve as confirmation of the diagnosis of acute pancreatitis.

Acute diverticulitis. Perforation of an acutely inflamed diverticulum of the sigmoid colon may engender symptoms and signs identical with pelvic appendicitis. Differentiation is practicable if an inflamed and thickened descending colon can be felt abdominally, or if a tender nodular mass is palpated rectally or bimanually. Nevertheless, the presence of peritonitis will usually make operation mandatory.

GYNÆCOLOGICAL CONDITIONS

Acute Salpingitis. The diathesis of the patient and the history of a previous attack, combined with discharge, paracervical tenderness, and thickening which is bilateral, are familiar diagnostic criteria. The pain is characteristically indicated 1 in. above the mid-point of the inguinal ligament. Extreme caution should, however, be observed in the diagnosis of right-sided salpingitis. Laparotomy in such cases has not infrequently shown a pelvic appendix and a Fallopian tube combined in one inflammatory mass. A pyosalpinx may extend up into the right iliac fossa resembling an appendix abscess. It will be found to be mobile and smooth when examined under an anæsthetic.

Torsion of the Fallopian tube, fimbrial and broad ligament cysts. These conditions are often indistinguishable from pelvic appendicitis, save by bimanual examination under anæsthesia.

Torsion of ovarian cysts and fibroids. Zachary Cope has stated that pain and vomiting are noted simultaneously in these two predicaments. Early diagnosis can be made when the smooth, tender, mobile pelvic swelling is felt soon after the onset of the attack, which is usually a sudden event, the time and circumstance of its occurrence being accurately described by the patient.

Ectopic Pregnancy. Tubal pregnancy is seldom diagnosed before leakage or rupture. These complications give rise to symptoms of lower abdominal pain and vaginal

bleeding. Shock and shoulder tip pain are inconstant. A history of amenorrhœa is not obtained in the majority of cases.

Delayed rupture of a Graafian follicle. Lower abdominal pain, tenderness and muscular guarding may be caused by delayed follicular rupture which produces intra-peritoneal hæmorrhage in the middle of the intermenstrual phase. In one such case seen by the author, the symptoms and signs were right-sided, with pyrexia, the abdomen contained over a pint of blood, and persistent bleeding was occurring from the left ovary.

URINARY DISEASES

Acute Pyelitis A high temperature, in the region of 103° F, rigors, vomiting, frequency, and pain in the right loin indicate pyelitis. Examination of the urine by the eye and nose should be supplemented by immediate microscopical scrutiny of one drop from a clean or a catheter specimen. The contrifuged deposit should then be examined. Proximity of an inflamed appendix to the ureter may produce small numbers of pus or red cells in the urine, but bacteria will not be present.

Ureteric Colic. Typical ureteric colic presents little difficulty in diagnosis. When, however, the pain is a fixed dull ache in the region of the cæcum, without radiation, remission or renal tenderness, confusion may arise until the urine is examined. The pain of renal and ureteric colic is more severe than can be produced by the appendix, the patient being incapacitated yet restless.

Hydronephrosis. Is also capable of producing prolonged severe pain in the right flank, with rigidity overlying an indefinite tender swelling. When the diagnosis is in doubt, an emergency intravenous pyelogram may be of help.

Pyonephrosis and Perinephric Abscess. When an abscess is formed secondary to acute or gross renal infection, the diagnosis from retrocæcal appendicitis is not difficult. The insidious perinephric infection which is sometimes seen in the aged, is more obscure having even fewer abdominal symptoms and signs than an inflamed appendix placed high behind the cæcum.

EXTRA ABDOMINAL DISEASE. Local inflammatory states outside, yet adjoining the peritoneal cavity, occasionally simulate the later stages of acute appendicitis.

Pleurisy and pneumonia. Basal right-sided pleurisy and right lower lobe pneumonia may both engender pain and rigidity in the right side of the abdomen. The rigidity is characteristically more pronounced in the upper than the lower quadrant. In the event of clinical and radiological examination confirming these diagnoses, it is wise to reconsider the history and review the abdominal signs. If these are still suggestive of appendicitis, operation is still indicated.

Psoas Abscess and Osteomyelitis of the Ileum may both give rise to an inflammatory mass in the right iliac fossa. Although not truly an inflammatory condition, a leaking atheromatous aneurysm of the right common iliac artery has been seen to produce pain, tenderness, and rigidity in the right iliac fossa exactly simulating an appendicitis. The patient's right leg was colder than the left.

General Medical Diseases. In addition to the two classical conditions of tabes dorsalis and diabetes which imitate acute abdominal states, herpes zoster, Bornholm disease, abdominal "migraine" and "epilepsy," should be included in the complete differential diagnosis of an obscure case.

The Treatment of Acute Appendicitis

Acute appendicitis should be treated by urgent operation in all cases excepting those in which a well-formed mass is present. An appendix mass is treated expectantly. So far as dogmatic generalizations have any place in the discussion of surgical treatment, this statement lays down the safest principles in the treatment of appendicitis, taking no account of the duration of the disease. It must, however, be modified and expanded to include the important factors of the patient's condition and the presence and extent of peritonitis.

(1) **Simple acute appendicitis (without perforation or peritonitis).** The appendix should be removed as soon as possible, irrespective of the duration of the disease. Except in special circumstances to be mentioned, this decision should not be influenced by thoughts that an early catarrhal inflammation might be aborted by chemotherapy, or that a "resolving" appendix may be treated conservatively.

(2) **Acute appendicitis with recent perforation and local peritonitis.** Early appendicectomy should be performed.

(3) **Acute appendicitis with an ill-defined or mobile mass and with local peritonitis.** If the condition of the patient is fair, examination under anæsthesia should be followed by exploration through a McBurney's incision. If it appears possible to remove the appendix without excessive trauma to surrounding structures, this should be done. Otherwise a separate stab drainage opening should be made, and a soft rubber drain laid down to the appendix, the exploratory wound being closed

(4) **Acute Appendicitis with General Peritonitis.** The treatment of the peritonitis must take precedence over that of the appendix. This will involve the administration of morphia, gastric intubation and suction, correction of dehydration and disturbed electrolyte balance, and chemotherapy. The position in which the patient is nursed is not of primary importance (*See* Peritonitis, treatment, Chapter IX, p. 332.) From this treatment there may result:

(*a*) Rapid resolution of peritonitis and rapid improvement of the patient's condition. Decision must then be taken as to whether removal of the appendix is desirable. Conservatism may be the safest course, but if localizing signs are still present in the right iliac fossa without any evidence of a mass, the patient will be benefited by appendicectomy.

(*b*) Resolution of the general peritonitis with localization in the right iliac fossa or pelvis Expectant treatment is continued and the abscess drained if it fails to diminish in size.

(*c*) Failure of improvement in the patient's condition. This calls for a review of the chemotherapy and intravenous therapy which is being pursued, and for intensification or modification of the treatment. Synergistic antibacterial agents may be employed, or more potent agents substituted A transfusion of fresh whole blood is often useful. Surgery is not indicated except for a patient whose belly is distended with pus, when simple drainage may effect a remarkable improvement.

(5) **Appendix Abscess.** An abscess involving the appendix is treated conservatively. Drainage is employed on certain definite indications. The principles of conservative treatment, as laid down by Ochsner and by Sherren and elaborated by MacNeill Love and Hamilton Bailey, are summarized as:

(*a*) No food or fluids by mouth. Nourishment was maintained in the original regime by small enemata. Intravenous therapy is now used.

(*b*) Nursing in the Fowler position.

(*c*) Gastric aspiration and lavage for vomiting and distension.

(*d*) Careful observation and chart recording by the nursing staff.

The adoption of these measures led to a significant decrease in mortality from appendix abscess and peritonitis between the wars. It has been argued that the advent of new antibiotics makes exploration and appendicectomy a safe procedure in all circumstances, and some excellent series of cases have been presented in support of this, both from America and in this country. It is felt, nevertheless, that the conservative attitude of mind to appendix abscess is not yet outmoded and the low morbidity and mortality from this method of treatment still support its continued use. The remarkable mortality previously associated with appendicectomy when the operation was performed between the 48th and 72nd hour has never been convincingly explained. Dehydration, toxæmia, and poor leucocyte response have been adduced as contributory causes. In more recent cases examined, however, no significant mortality or morbidity has been noted from operation, whatever the duration in time of the disease.

It is therefore felt that each individual case of appendicitis should be assessed clinically rather than chronologically and that the degree of localization of the abscess is a safer guide to treatment than the clock. A regime which may be adopted in the treatment of appendix abscess is as follows:

(1) The treatment proposed is explained to the patient and relations, or parents. The patient is nursed in a comfortable semi-sitting position.

(2) The pulse is taken half-hourly, and the temperature four-hourly. The size of the abscess may be outlined on the abdominal wall if of assistance to those in charge of the case.

(3) A Ryle's tube is passed, and the stomach contents aspirated. No fluids are given by mouth save for small quantities of water or fruit juice which are then aspirated back.

(4) An intravenous drip is set up, and the patient's basic daily fluid intake is planned in advance.

(5) Small doses of morphia (gr. $\frac{1}{6}$–$\frac{1}{8}$) or pethidine 25–50 mg. may be given, but should not be administered so frequently as entirely to obscure the patient's symptoms. Necessity for continued analgesics indicates that the inflammatory process is not being controlled by conservative treatment.

(6) Antibiotics should be withheld *initially* unless there is evidence of toxæmia or of peritonitis beyond the confines of the mass. This therapeutic restraint is of value in assessing the efficacy of treatment. If indications for drainage arise, parenteral antibiotics should be given before operation. Their routine administration after the first few hours as an adjunct to conservative treatment is a matter for individual choice. Although theoretically desirable, in practice a rigid walled abscess which fails to resolve has sometimes been produced and drainage has been delayed until a lesion of carcinomatous consistency has to be dealt with by excision

Results of Conservative Treatment. 80–85 per cent of abscesses treated conservatively will resolve and be suitable for interval appendicectomy in three months' time.

Indications for Drainage of an Appendix Abscess

(1) Spread of peritonitis. Spread of peritonitis, despite energetic conservative treatment, is a classical indication for drainage. It must, however, be emphasized that if the patient is gravely ill, even a minor intervention may well prejudice recovery so that operative treatment must be considered at the first sign of diffusion of the infection.

(2) Persistence of, or increase in, pain should call for reconsideration of conservative measures.

(3) An abscess which persists in its original size, enlarges, or becomes more tense or tender, should be drained without waiting for signs of œdema of the overlying wall. Delineation of the swelling's dimensions on the patient's skin is a useful guide to change in size, and serial white cell counts which give some guide to the activity of the infection.

(4) Increasing toxæmia, formerly considered a signal for intervention, should be regarded with reserve. Common causes of this state are errors in electrolyte balance, inadequate exhibition of antibiotics, and the presence of pus in more dangerous regions such as the sub-diaphragmatic areas.

Method of drainage. The incision is made over the most prominent part of the swelling. It should be large enough to admit two fingers and should, if possible, split the muscles rather than cut them in the first place. In abscesses lying well laterally in the right iliac fossa, the operator may aim to enter the abscess from its right border and will thus avoid transgressing the main peritoneal cavity. When the internal oblique muscle has been penetrated by blunt dissection, the finger is used to enter the abscess cavity. The abscess should be evacuated with the sucker and explored with the finger for necrotic material, fæcaliths, and foreign bodies A corrugated rubber drain is then laid into the abscess and the abdominal wall loosely sutured with interrupted stitches.

The pelvic abscess It is desirable to allow a pelvic abscess to point and discharge through the vagina or rectum, and this is more likely to happen if antibiotics are withheld. In the event of surgical drainage being necessary, use of the finger is preferred to forceps, however blunt these may be. A persistent high placed abscess will have to be treated by anterior abdominal exploration, the roof of the cavity being removed and small gut allowed to fill the dead space created.

The Management of Appendicitis in Special Circumstances

Pregnancy. The diagnosis is sometimes difficult; degeneration of a fibroid, concealed accidental hæmorrhage, and acute pyelitis being three of the more easily confused conditions. Once the diagnosis is established there can be little debate concerning the best treatment, which is early operation The risks of abortion increase with the time for which an acutely inflamed organ is left in the peritoneal cavity, and future fertility may also be impaired. In circumstances in which the surgeon is confident that the inflammation is sub-acute or non-obstructive, a case may very rarely be made for antibiotic treatment in hospital, and observation. A good recent review of the subject has been made by R. B Parker (1954).

Infants and Children. It has already been stated that the diagnosis of appendicitis should not be discarded on account of a concomitant respiratory infection, "gastro-enteritis" or even one of the exanthemata Though the short omentum of the child has been adduced as a principal reason for the indifferent or delayed walling-off of the

infection, a more potent factor is the poor peritoneal response of infancy. This makes urgent operation essential in nearly all cases. In the rare instance in which a well-formed mass is present, it should be treated conservatively, but with trepidation, as intraperitoneal rupture is not unknown

THE TROPICS Appendicitis in the tropics pursues a rapid and fulminating course, diarrhœa being a frequent symptom, and confusing the diagnostician with thoughts of bacillary and amœbic dysentery, both conditions sometimes masking or simulating appendicitis. If appendicitis is strongly suspected by reason of the local signs, and if stool examinations cannot be performed rapidly, exploration should be done, antibiotics effective against entamœba histolytica being administered pre- and post-operatively. The best single drug for this purpose is aureomycin. Emetine, penicillin, and streptomycin are a useful combination. A smear of the contents should be made as soon as the appendix is removed.

AT SEA AND IN UNSUITABLE SURGICAL CONDITIONS. The surgeon in these cases has severe responsibilities He first must decide whether the appendix is obstructed and liable to perforate, or whether the inflammation is sub-acute or catarrhal in character. If in doubt, a re-assessment of the patient after a rest of two hours will often give an indication of the course which the attack is pursuing. If the appendix is acutely inflamed and obstructed it should be removed, provided that surgery and anæsthesia are of a *reasonable standard of skill.* In war the tactical situation must also be assessed, as a conscious patient with an intact abdomen travels or swims with more chance of survival. Should the inflammation be deemed sub-acute or catarrhal, if a mass is present, or if facilities are not of a standard fair to the patient, antibiotics, suction, and intravenous fluids should be used.

Pre-operative Treatment and Investigations. The patient should be operated upon when his condition is at its best. This indicates urgency in an acute, but unruptured appendix and purposeful delay in a patient whose peritonitis has the upper hand. Though the minimum of disturbance is the best preparation for any operation, certain essential measures must be taken before the theatre is reached.

(1) The urine must be examined, preferably by the microscope, as well as by the routine qualitative ward tests (which should include assessment of urinary chlorides in an ill patient)

(2) Clinical examination of the chest should be assisted by a portable X-ray film if there appears any doubt as to the findings

(3) A Ryle's or duodenal tube should be passed and the stomach aspirated. This is a wise precaution even in the absence of vomiting, ileus or a recent meal.

(4) If the patient is dehydrated, intravenous fluids should be administered. When dehydration is not gross, this measure may be postponed till the operation has begun.

(5) Parenteral chemotherapy should be instituted if peritonitis is present. The absorption of drugs instilled down the Ryle's tube is capricious, and sulphonamides, though effective against bowel organisms are not suitable for parenteral use in patients whose urinary output and reaction are dubious.

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Chloramphenicol, a potent anti-peritonitic drug, has to be administered by mouth, and the incidence of agranulocytosis reported from its use, though small, has decreased its popularity.

(6) Shaving, skin preparation, and catheterization may be humanely omitted till the anæsthetic has been started.

Choice of anæsthetic. Though no longer the privilege of the surgeon, it may sometimes fall to one's lot to advise a junior colleague, and the author's preference is for pentothal, nitrous oxide and oxygen, supplemented by muscle relaxants if difficulties are encountered. Endotracheal intubation is an additional safeguard against copious vomiting, even if a Ryle's tube is already in position. Cyclopropane is an excellent emergency anæsthetic agent, provided that the theatre humidity is reasonable. In less civilized circumstances, ether given by the Nuffield vaporizer or by the open method gives a satisfactory anæsthetic. There is seldom any indication for the use of a local anæsthetic, and unless the operator is greatly experienced in the induction of spinal anæsthetics, this method is better avoided.

Instruments. The appendix can, and has been, removed with the minimum of instruments, including domestic cutlery. The general laparotomy set provided in hospital theatres is more than adequate, but should always be supplemented by deep retractors of the gall bladder or Lloyd Davies pelvic type, and by long curved Spencer Wells or Moynihan forceps.

Operation for Removal of an Acutely Inflamed Appendix. When placing the patient on the table, it must be ensured that the Trendelenburg position can be assumed if necessary. In female patients a catheter should be passed if this has not already been done. Preparation of the abdomen may be preceded by an abdominal and pelvic examination, if an indefinite mass is still suspected, or if an alternative pelvic condition is under consideration. This examination may also influence the site of the incision.

Choice of Incision. The majority of surgeons favour McBurney's muscle-splitting incision It has the advantage of a direct approach to 80 per cent of appendices, without disturbance of the general peritoneal cavity, and extension into the loin, or across the sheaths of the right and left rectus muscles to expose the pelvis is easy. The paramedian incision is reserved for cases of pelvic appendicitis, or for cases in which pelvic pathology may or may not be due to the appendix. A modification of Battle's incision, by which the rectus sheath is opened transversely, the muscle retracted medially, and the nerves and vessels identified before the posterior layer is divided transversely, is sometimes useful in a child, or in a patient whose appendix lies in relation to the terminal ileum.

Site of Incision (McBurney) This should overlie the estimated position of the appendix and will also vary with the individual contour of the abdomen. It will be placed over or above the line joining the anterior superior iliac spine to the umbilicus when the appendix is retro-cæcal, and below this line in other cases. The skin incision is best made almost horizontal, parallel with the lower abdominal skin crease. It should be generous in extent, for there is no place for "tiny scars" in operating on an acute appendix The object is to secure such a good exposure that the whole operation can be conducted in comfort and safety under direct vision. The external oblique muscle is widely divided in the line of its aponeurosis, the division being continued into the upper fleshy part of the muscle. Retraction then displays the sheath and transverse fibres of the internal oblique muscle. This muscle may be seen to have a superficial and a deep layer,

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that it is unwise to press home his attack at all costs, drainage should be employed. Removal by the retrograde method may be practised when the appendix lies retrocæcally, but does not relieve the surgeon from the obligation to have the whole of the organ under direct vision. An inspection of the appendix bed for hæmorrhage must always be made before starting to close the abdomen, as retraction of the cæcum may dislodge a ligature from the meso-appendix. Finally, if the appendix is perforated, the operator should assure himself that no fragment of the organ or fæcolith remains in the peritoneal cavity.

Drainage of the Abdomen. It may be unfashionable to assert, against those who regard drainage as a mediæval measure, that no harm has yet been seen from it, and that some good appears sometimes to have been done. A good statistical case has been made against drainage by Moloney, Russell, and Wilson (1950) in their excellent and comprehensive article on appendicitis, and they are in exalted and numerous company. Few surgeons would now employ drainage as a routine, but it is still performed by the author on certain definite, if infrequent, indications. If there is gross purulent or fæcal contamination of the appendix bed, persistent capillary oozing, friability or acute inflammation of the cæcum, the remnants of an appendix abscess present, or if the appendix cannot easily be removed, a slip of soft corrugated rubber, Penrose tubing or Paul's tubing should be laid in the area and brought out through a separate stab incision in the loin. This should be shortened daily and removed on the fourth day.

Intra-peritoneal chemotherapy. The institution of intra-peritoneal chemotherapy in the sulphonamide-penicillin era was an advance which diminished the mortality of appendicitis with peritonitis (R. Vaughan Hudson, Rodney Smith, 1942), though this was not confirmed by other writers The use of intra-peritoneal drugs is not now so general since the introduction of agents potent against bowel organisms which can be administered parenterally. Nevertheless, a light dusting of the appendix bed with penicillin and sulphanilamide powder is still thought by many surgeons to be of service and is not likely to give rise to complications of local irritation, overgrowth of contaminating organisms, or anuria, if the quantities used are small. The only danger is that which may arise from sensitivity of the patient to these drugs.

Closure of Abdomen. The cæcum and terminal ileum are first replaced with gentleness in their correct anatomical position. The peritoneum is then closed with continuous 0 chromic catgut, the transversus, internal oblique and external oblique muscles by interrupted 0 chromic catgut sutures. It is important that the transversus and deep layer of the internal oblique muscles be included in the sutures, and this may be facilitated in a deep wound by a J shaped needle. These stitches should not be tightly tied. If the lateral border of the rectus sheath has been divided, particular care must be taken in its repair as it constitutes one of the pillars of the abdominal wall. In the event of the wound being grossly contaminated, the muscular layers may be drained by a slip of soft rubber, and it need hardly be stated that the hæmostasis is of the utmost importance when infection is present.

Histological Examination. Every appendix removed should be submitted for histology. A perforated appendix should be aseptically swabbed and the sensitivity of organisms determined if the operator has omitted to secure a sample of the peritoneal exudate.

Post-operative Care. During the operation for acute appendicitis it is recommended that an intravenous drip be set up, if this has not been done before. A short bevelled 1 or 12 needle is inserted by the anæsthetist into one of the veins of the distal forearm

the latter being closely applied to the tough fibres of the aponeurosis of the transversalis muscle. It is divided by blunt dissection in the line of its fasciculi, a watch being kept for segmental nerve fibres. The transversalis muscle fibres may need additional attention before the surface of the peritoneum is exposed, and digital stretching of this layer of the incision assists exposure.

Intra-abdominal Procedure. On opening the peritoneum, escape of gas or the presence, appearance, and odour of free fluid should be noted. If the fluid is turbid, a specimen should be taken for culture, and for evaluation of the organism's sensitivity to antibiotics Retractors are inserted and a visual examination of the peritoneum and its contents is carried out. All gloves are washed and a gentle palpation is then performed. This will determine the condition and position of the appendix. If it appears difficult to remove through the incision made, although muscular relaxation is adequate, a formal extension of the wound should be made at this stage, before traumatic manipulations have been essayed If location of the appendix is difficult, the cæcum is identified and its tæniæ followed down to their point of convergence. Denis Browne's dissecting forceps give rise to the least trauma in handling the intestine in situations where the fingers cannot be employed. When the appendix is found to lie retrocæcally, the cæcum must be delivered upwards out of the wound, division of the peritoneal folds at its base being sometimes necessary.

Removal of the Appendix. The tip of the appendix is grasped by tissue forceps. Whenever possible, the two main vascular leashes in the appendix mesentery should be ligated individually with 0 chromic catgut on an aneurysm needle. When the meso-appendix is thickened, œdematous and friable, the serial application of curved artery forceps with division of the mesentery close to the appendix is preferred: each section of mesentery grasped by forceps is then ligated Whilst this is being done, the assistant should protect the terminal ileum from injury. When the mesoappendix has been divided up to the cæcal wall and complete hæmostasis secured, a serosal purse-string suture is passed on an atraumatic needle in a circle 1 cm from the base of the appendix. If the cæcum is œdematous, this step is omitted. The base of the appendix is crushed and a ligature of 1 chromic catgut is tied just proximal to the crushed segment. The stump of the appendix may be carbolized if the operator is convinced of the necessity of this precaution. (Crude carbolic acid is such a dangerous liquid that the author feels it should not be allowed in the operating theatre) The stump is then invaginated and the purse-string suture tied. Alternative methods of invagination employed if the cæcum is œdematous are the "Z stitch" and the drawing across of residual mesoappendix to cover the area, or a three-layer closure similar to that performed for the duodenum in gastrectomy. The merits and demerits of stump invagination have been exhaustively discussed—the incidence of intramural abscess, sloughing of the non-invaginated stump, and adhesions to the non-invaginated stump appear approximately equal. Gangrene of the appendix involving the cæcal wall may usually be surmounted by invaginating the whole base of the cæcum. If intramural abscesses are present, the butt end of the cæcum may be resected and closed It is on very rare occasions necessary to sew a catheter into the cæcum, or exteriorize it into the wound if it appears in danger of sloughing

Difficulty in removing the Appendix When the appendix is firmly embedded in inflammatory tissue or forms part of the wall of an abscess, the surgeon must decide if he can safely remove it without excessive local tissue destruction and trauma. If he feels

disaster and exploration must be performed as soon as the patient can be made fit to be taken to the theatre.

PARALYTIC ILEUS. Ileus may persist for some days after operation. Continuance of gastric suction and intravenous therapy is called for, with special attention to potassium balance. The ileus following appendicular peritonitis is almost invariably due to persistence of infection, and chemotherapy should be continued, or more potent agents used. (*See* Peritonitis, Chapter IX, Treatment of Paralytic Ileus.)

The onset of obstruction following ileus is heralded by resumption of vigorous bowel sounds, colicky pain, visible peristalsis, and increased aspirations. Though the use of a Miller-Abbot tube may circumvent a sub-acute obstruction, the more severe case demands exploration.

INTRA-PERITONEAL ABSCESSES. The formation of discrete localizations of pus in the abdomen is manifested by the slow progress of the patient, persistent pyrexia, and a high white cell count. A disadvantage of treatment by antibiotics lies in the suppression of fever and leucocytosis, allowing reaction to continue around collections of pus which are sterile, or of a low grade infection. This calls for increased vigilance on the part of the surgeon and for routine abdominal and pelvic examinations during convalescence. The majority of these collections will resolve without drainage on rest in bed, local heat, and the vis medicatrix naturæ. Chemotherapy should not be continued indefinitely, but reserved in case it should be necessary as an insurance during a drainage operation.

PYLEPHLEBITIS AND PORTAL PYÆMIA. Spread of infection from the ileo-colic vein to the liver is now extremely rare, the presenting features of pyrexia, rigors, and pain in the right hypochondrium being more commonly due to coincident pyelitis, or to sub-phrenic suppuration. The treatment is by intensive chemotherapy rather than operation.

DEEP VEIN THROMBOSIS Patients convalescing from acute appendicitis are no more immune than other surgical cases to this complication. The benefits which accrue from the comparative youth of those subjected to appendicectomy, and their short stay in bed, is offset by the increased incidence of deep vein thrombosis associated with peritonitis. It is not the general practice in this country to treat emergency abdominal patients with prophylactic anti-coagulants. Nevertheless, it may be justifiable to select certain patients by reason of age, severity of peritoneal infection, obesity, or probable long stay in bed, and exhibit to them Heparin 5,000 units b d., or Dindevan 50 mg. b.d., as a prophylactic measure, from the second post-operative day. It is in the hands of the nursing staff that the early detection of deep vein thrombosis lies Patients will inform them of minor leg discomforts with which they would be unwilling to worry their doctor. This does not excuse the ritual examination of the calves and legs of surgical patients by the medical staff. When a clinical suspicion of calf vein thrombosis arises, full therapeutic anti-coagulant therapy should be instituted without awaiting more definite signs.

Death Rate. Rodney Maingot, in his authoritative chapter on Acute Appendicitis, estimates the average fatality rate for acute appendicitis without perforation throughout Great Britain and America at 0·3 per cent. Including all types of cases of abscess and peritonitis, the death rate varies from 1–5 per cent. The value of these figures is questionable since appendicitis is a disease with remarkable variations in individuals and in different areas. Statistics will obviously be better when culled from large surgical centres in towns where rapid admission and urgent operation are easy, than from the more remote fastnesses of Wales and Scotland when the higher mortality is due to delay in calling

or dorsum of the hand. A young adult patient with acute appendicitis should receive two litres of 0·18 per cent sodium chloride combined with 4·3 per cent glucose and water (Isotonic ⅕ normal saline and glucose) during and after the operation, after which the needle should be withdrawn. Should vomiting have been severe, dehydration evident, or peritonitis diffuse, this quantity will be increased. (*See* Peritonitis, Chapter IX, p. 333.)

Though this routine administration of intravenous fluids may seem an unnecessary interference, the benefit to the patient is manifest on the first post-operative day. Thirst is not present, the patient looks well, and there is no necessity to force oral fluids into an atonic stomach and intestine which is loth to receive them. When the patient is conscious, a comfortable semi-upright position should be assumed and sips of water are allowed, as requested. The gastric tube may be removed unless peritonitis was observed at operation. In such cases it must remain in position and aspirations are conducted at hourly intervals. In the first 24 hours following operation only a modest quantity of mixed fluids (2–3 pints) should be administered by mouth unless the patient, by reason of his flat abdomen, normal peristalsis, absence of nausea, and passage of flatus, is obviously able to accept more. The peritonitic patient will, of course, receive all his fluids by intravenous infusion. As soon as normal bowel sounds are heard and a good flatus result is obtained by a rectal tube, normal feeding may be cautiously resumed. No aperients or an enema larger than 4 oz. should be permitted for the first five days following operation The patient is normally allowed to sit out of bed on the second day and is relieved of his skin stitches on the seventh day after operation. Before discharge from hospital a careful examination of wound, abdominal wall, right iliac fossa, and pelvis must be made.

Complications of Appendicectomy. Apart from the multitudinous untoward events which occasionally follow upon or coincide with any abdominal operation, removal of a gangrenous or perforated appendix may be attended by its individual notorious sequelæ. (A brief list is appended with even shorter notes on treatment.) It cannot be emphasized too strongly that complications must be anticipated with abnormal foresight and prevented or treated with the determination of a military commander for the highest *standards of post-operative surgical care to be achieved*

THE WOUND. An hæmatoma should be evacuated as soon as it is discovered. This is more thoroughly done in the theatre than in the ward. Infection will demand the cutting of stitches, drainage, and bacteriological culture. Apparent discharge of "fæces" is more often due to anærobic infection of a wound hæmatoma than to true fistula formation. A true fæcal fistula will close unless unsuspected pathology in the ileo-cæcal area is the cause Persistence of a fistula calls for reinvestigation of the patient which should include a biopsy curettage of the track. If conservative measures, including the application of silver nitrate fused to a probe are not successful, an elective operation for closure will be necessary Wound breakdown is almost unknown from McBurney's incision It is heralded in a paramedian wound by a profuse serous discharge which should always call for immediate investigation, followed by re-suture.

INTRA-PERITONEAL COMPLICATIONS Spreading peritonitis is still sometimes seen in spite of the antibiotics. *If the onset is insidious and the operator is satisfied by the condition* of the cæcum at operation and his stump closure, the treatment will be by general measures directed against peritonitis and by a powerful antibiotic agent such as intravenous terramycin. A sudden onset of peritonitis rapidly involving the abdomen indicates

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Pylephlebitis and Portal Pyæmia. Spread of infection from the ileo-colic vein to the liver is now extremely rare, the presenting features of pyrexia, rigors, and pain in the right hypochondrium being more commonly due to coincident pyelitis, or to sub-phrenic suppuration. The treatment is by intensive chemotherapy rather than operation.

Deep Vein Thrombosis. Patients convalescing from acute appendicitis are no more immune than other surgical cases to this complication. The benefits which accrue from the comparative youth of those subjected to appendicectomy, and their short stay in bed, is offset by the increased incidence of deep vein thrombosis associated with peritonitis It is not the general practice in this country to treat emergency abdominal patients with prophylactic anti-coagulants. Nevertheless, it may be justifiable to select certain patients by reason of age, severity of peritoneal infection, obesity, or probable long stay in bed, and exhibit to them Heparin 5,000 units b.d., or Dindevan 50 mg. b.d., as a prophylactic measure, from the second post-operative day. It is in the hands of the nursing staff that the early detection of deep vein thrombosis lies. Patients will inform them of minor leg discomforts with which they would be unwilling to worry their doctor. This does not excuse the ritual examination of the calves and legs of surgical patients by the medical staff. When a clinical suspicion of calf vein thrombosis arises, full therapeutic anti-coagulant therapy should be instituted without awaiting more definite signs.

Death Rate. Rodney Maingot, in his authoritative chapter on Acute Appendicitis, estimates the average fatality rate for acute appendicitis without perforation throughout Great Britain and America at 0 3 per cent. Including all types of cases of abscess and peritonitis, the death rate varies from 1–5 per cent. The value of these figures is questionable since appendicitis is a disease with remarkable variations in individuals and in different areas. Statistics will obviously be better when culled from large surgical centres in towns where rapid admission and urgent operation are easy, than from the more remote fastnesses of Wales and Scotland when the higher mortality is due to delay in calling

the doctor and even sometimes to refusal to enter hospital. Providing that sound surgical principles have been followed and that post-operative care has been assiduous, no surgeon need feel unduly distressed by two fatalities in every hundred of his cases.

"CHRONIC APPENDICITIS"

Chronic appendicitis is a loose clinical term which includes four conditions sufficiently distinct to be classified by their pathology.

(1) Sub-acute appendicitis.
(2) Recurrent appendicitis.
(3) True chronic obliterative appendicitis.
(4) Mucocœle.

SUB-ACUTE APPENDICITIS. This presents as a vague central pain accompanied by disinclination for food, or nausea, but rarely by vomiting. The pain may localize in the right iliac fossa, but more commonly is unremarked till tenderness is elicited in this area by the examiner. There is a slight rise in temperature, the pulse rate remains normal and little constitutional disturbance is observed. The history of a previous attack is sometimes given and the present attack has frequently been preceded by a sore throat or mild enteritis. Although there is every probability of resolution, appendicectomy is the right treatment in these cases. Treated conservatively and discharged for out-patient observation, recurrence is probable with re-admission in emergency at a time or in a place equally inconvenient for patient and for surgeon, and with symptoms more typical of obstructive appendicitis.

Macroscopically, the organ appears slightly swollen and pink. Its consistency is firmer than the normal. Small glands may be seen in the appendix mesentery, or adjoining the terminal ileum. Microscopically, early mucosal and submucosal inflammation is found, with cellular infiltration of these layers, and evidence of lymphoid hyperplasia.

RECURRENT APPENDICITIS. Recurring attacks of central abdominal colic, without fever, associated with tenderness over the appendix, are characteristic of this variety of chronic appendicitis. Some patients also complain of dyspepsia, flatulence, fat intolerance, irregular bowel action and upper abdominal pain (the so-called "appendicular dyspepsia"). The underlying pathology is a partial appendicular obstruction due to angulation or previous sub-mucosal fibrosis, past which the appendix is attempting to expel a fæcal concretion, inspissated contents, or even worms or a foreign body. Diagnosis is made by an exhaustive history and, if possible, by observation during an attack, with the elicitation of tenderness over the appendix. Cæcal distension, small bowel colic and chronic disease of the ileo-cæcal area must be excluded.

X-ray Diagnosis. The value of X-ray investigations in the diagnosis of chronic appendicitis is doubtful. A barium meal may assist in excluding disease of the terminal ileum, and a Barium Enema may perform a similar service for the cæcum and ascending colon. It is rarely justifiable to request these examinations in suspected chronic appendicitis. The Barium Magnesium Sulphate Meal may show any of five features, namely, failure to fill, delay in emptying, abnormal position and kinking of the appendix, tenderness over the outlined appendix and the presence of fæcaliths or foreign bodies

Osborne (1953) has concluded by the comparison of a series of patients undergoing appendicectomy on X-ray criteria, with a series of patients whose appendices were

removed incidentally to a gynæcological operation following X-ray examination, that there is no value in the procedure. Nevertheless, the support of a clinical diagnosis by the demonstration of filling defects in an appendix which fails to empty, has been found of assistance by the author.

Treatment of Recurrent Appendicitis. Appendicectomy should be performed if the diagnosis is reasonably firm. If doubt exists, a period of observation by periodic interview and examination is instituted. Bowel function may be corrected by dietary measures, or if these fail, by a physiological bulk aperient such as Normacol or Isogel. Antispasmodics should initially be avoided unless the functional aspects of the clinical picture seem to outweigh the organic. About 50 per cent of patients observed for dubious symptoms will eventually qualify for appendicectomy and the majority of these will be relieved of their symptoms by operation.

TRUE CHRONIC AND OBLITERATIVE APPENDICITIS. This condition is comparatively rare. The process is one of distal low grade inflammation which spreads proximally along the mucosa and submucosa, destroying the mucosal lining and obliterating the lumen. A shrivelled cord-like structure is produced, unless residual mucous glands remaining in the distal part begin to secrete, giving rise to mucocœle. Obliterative appendicitis does not often cause typical symptoms which lead to its removal and is frequently found incidentally at post-mortem. When symptoms occur they are of the recurrent type and tenderness may be found on examination.

MUCOCŒLE (GLOBULAR MYXOMA). The formation of a mucocœle is an uncommon sequel to obstruction of an appendix, the lumen of which is sterile. Two main varieties are described. In the first, the epithelial mucus secreting cells are in a single layer. In the second, papilliferous overgrowth of these cells is seen, in some cases to a carcinomatous degree. (A recent short review of two cases and the literature has been written by J. H. Johnston) Escape of mucus secreting cells will lead to pseudomyxoma peritonei, which may be benign or malignant in character. Mucocœle most commonly presents as a symptomless swelling in the right iliac fossa or pelvis, or is discovered incidentally at operation. A history of occasional pain in the right iliac fossa, or of an attack of appendicitis is sometimes to be obtained.

Notes on Chronic Appendicitis

Diagnosis. The evaluation of vague symptoms in the right iliac fossa is no easy surgical task. Exclusion of the original organic disease, particularly of the pelvis, must be completed without excessive expenditure of time and money in the radiological department. The two functional conditions which most commonly simulate appendicitis in young women are cæcal distension combined with incoordinated large bowel function, and the ovarian disorders of delayed rupture of a Graafian follicle, or pre-menstrual ovarian congestion. If the symptoms are not reasonably compatible with chronic appendicitis, a period of out-patient observation is preferable to exploratory surgery. When, however, appendicectomy is indicated, it should be explained to the patient that the diagnosis is not a concrete fact, but a probability.

The Operation for Chronic Appendicitis. An examination of the pelvis under anæsthesia may first be conducted. McBurney's approach is used. The skin incision may be as small as the artistic feelings of the surgeon dictate, provided that the deeper layers of the abdominal wall are opened sufficiently freely to see the appendix, cæcum and

terminal ileum, and that two fingers at least can be inserted into the abdomen to feel the pelvic contents and gall bladder.

Pathology. All chronic appendices removed should be examined microscopically and the histology recorded. It must be stated that many organs removed for chronic appendicitis show a diversity of feature sometimes not far removed from the normal and that the histological report may be found to vary with the benevolent disposition of the pathologist.

A final note must be made on the work of Masson who found overgrowth of the sub-mucosal nerve plexus and increase in numbers and size of ganglia in specimens of chronic appendicitis, which might explain the production of symptoms by these organs. Complete confirmation of these changes has not rewarded the efforts of other recent workers, the myenteric plexus also appearing anomalous in control series of normal appendices.

OTHER PATHOLOGICAL CONDITIONS OF THE APPENDIX

Actinomycosis and tuberculosis have both been recorded as affecting the appendix without other obvious gastro-intestinal lesions. If these diseases are associated with acute inflammation, macroscopical diagnosis is unlikely and recognition will only result with microscopy. Prompt institution of the appropriate antibiotic treatment will avoid a probable fæcal fistula.

INTUSSUSCEPTION. Intussusception of the appendix occurs in children later than ileal and colic intussusception, the common age group being between two-and-a-half and ten. It is predisposed to by the fœtal type of cæcum and appendix. The symptoms and signs resemble acute appendicitis so closely that the diagnosis is usually an operative one. An excellent recent review of this condition has been published by Forshall (1953).

CARCINOMATA OF THE APPENDIX. Three types of malignant neoplasm are recorded arising in the appendix:

Carcinoid.
Adenocarcinoma.
Lymphosarcoma

All are rare, the carcinoid being the most common.

Carcinoid (Argentaffinoma) The incidence of carcinoid was originally assessed at approximately one in a thousand appendices removed at operation. The present incidence would seem in the range of 0·2 per cent to 1 per cent. A carcinoid is seen as a small hard yellow nodule in the sub-mucosa, commonly near the tip of the organ. It is sometimes palpated and recognized at operation, but more often found on post-operative examination of the specimen The origin of the carcinoid was formerly believed to be in Auerbach's plexus. Masson's theory of endodermal origin from the Kultschitzky cells associated with the crypts of Lieberkuhn is now widely accepted. Carcinoid of the appendix does not metastasize so frequently as carcinoid of the terminal ileum, and recurrence is rare following appendicectomy If recognized at operation, a careful search for mesenteric glandular metastases should be made, and a more radical procedure contemplated if these are present.

Adenocarcinoma of the Appendix arises near the base of the appendix, sometimes being confined to the organ itself, but more often involving the cæcum. Diagnosis by

appearance and feel is possible unless an inflammatory condition is also present. The decision for hemicolectomy is then best made by a frozen microscopical section. Three sub-divisions of the adenocarcinoma in or involving the appendix may be made: papilliferous, with or without mucocœle, ulcerating, and stenosing.

Lymphosarcomata are exceptionally rare and arise from the lymphoid follicles of the appendix. A case simulating chronic appendicitis with a brief review has been published by Henley and Slack (1954).

References

Aird, I. (1949) *Comp. Surg. Studies*. 705. *Livingstone*, Edinburgh
Annotation (1953) *Brit. Med. J.* 2, 1148.

Forshall, I. (1953) *Brit. J. Surg.* Vol. XL. 305.
Henley, F. A. and Slack, W. W. (1954) *Brit. Med. J.* 1, 378.
Hudson, R. V. and Smith, R. (1942) *Lancet*, 1, 437.
Johnston, J. H. (1954) *Brit. Med. J.* 1, 135.

rofts, Inc. New York.

Moloney, G. E., Russell, W. T. and Wilson, D. C. (1950) *Brit. J. Surg.* Vol. XXXVIII, 52.
Osborne, G. (1953) Cambridge M.D. Thesis.
Reimann, S. P. (1918) *Amer. J. Med Sci.* 156, 190.
Parker, R. B. (1954) *Lancet*, 1, 1252.
Shepherd J. A. (1954) *Lancet*, 2, 299.
Sworn, B. R. and Fitzgibbon, G. M (1932) *Brit. J. Surg.* 19, 410.
Tagart, R. E. B. (1953) *Brit. J. Surg.* Vol. XL, 437.

Woodruff, R. and McDonald, J. R. (1940) *Surg. Gynec. Obstet.* 71, 750.

CHAPTER IX

PERITONITIS

J. H. LEES FERGUSON

Historical Note. It is salutary to reflect upon the remarkable changes which have attended the course and outcome of peritonitis during the last few years. The advent of antibiotics, and the better understanding of fluid and electrolyte disturbance, have so modified the natural history of peritoneal inflammation that many of the younger generation of surgeons may fail to appreciate the grave anxieties which they are now spared. The reader may be referred to Mr. Sampson Handley's classical Hunterian lecture.

Incidence and Mortality. The incidence of peritonitis is impossible to assess except by approximation of the numbers of cases seen in large general surgical centres serving a fixed population. The Registrar General's figure of deaths from peritonitis in 1950 was 107. The number of cases in which peritonitis contributed to death must be far in excess of this.

ANATOMY OF THE PERITONEAL CAVITY. The minutiæ of the anatomy of the peritoneal cavity are not of practical importance in relation to peritonitis. It is, however, usual to consider the general conformation of the peritoneum and its contents, inasmuch as some explanation may thereby be afforded of the routes of spread and sites of collection of infected peritoneal effusions.

When the body is supine, the vertebral column and great vessels form a longitudinal barrier which theoretically localizes fluid to the right or left of the abdomen. These paravertebral spaces are again divided longitudinally, on the right side by the cæcum, ascending colon and mesentery, and on the left by the descending colon and mesentery, forming the paravertebral and paracolic gutters. Fluid in these channels may therefore gravitate downwards into the pelvic sump, or upward toward the diaphragm.

The transverse colon, mesocolon, and great omentum form a transverse barrier dividing the upper abdomen from the remainder of the peritoneal cavity, and fluid passing this obstacle will often do so by the paracolic route. Mitchell, by his studies of barium injections into infant cadavers sets forth interesting and original views on intraperitoneal spread of effusions

(The subhepatic and subdiaphragmatic spaces will be considered in conjunction with subphrenic abscess)

The importance of these anatomical divisions is often overshadowed in practice by the volume of the exudate, the position and movement of the patient, by mobility of the gut and by the presence of gas in the peritoneal cavity, which fortifies the effects of gravity on intraperitoneal fluid.

PHYSIOLOGY. The normal peritoneum functions as a dialysing membrane in the secretion and absorption of peritoneal fluid. This process is mainly vascular, the lacteals playing little part in removal of fluid Particulate matter introduced into the peritoneum appears to collect in greater concentration around the great omentum, the roots of the mesenteries, and the diaphragm.

LOCAL PATHOLOGY. The lining cells of the peritoneum respond to inflammation by swelling and desquamation; the underlying tissues by hyperæmia and rapid secretion of exudate from dilated capillaries. This is at first thin and clear, becoming turbid as its leucocyte content rises, and is finally thick and purulent. Microscopic examination of the exudate will at first show bacteria and polymorphonuclear leucocytes. Wilkie observed that persistence of organisms and degeneration of pus cells after some hours of infection indicated a poor prognosis, whereas phagocytosis and disappearance of bacteria was an indication of a successful outcome. It is notable that, on culture, the pus is not infrequently sterile, even in the early stages of infection.

The surface of the peritoneum is red and dull, and œdematous. It becomes covered by deposition of fibrin, which appears initially as flakes, and later builds up into a friable layer, yellow-white in colour, and several millimetres in thickness.

Local intestinal movement is repressed by direct inhibition of the myenteric plexus, and the adjoining peritoneal surfaces become matted together by the adhesive nature of the fibrinous deposit The omentum may be involved in this stage of primary localization. If the infection is overcome, all evidence of local tissue reaction will disappear, a process known as resolution. If the infection persists, but remains localized, an abscess will result.

In certain circumstances not fully understood, the fibrinous exudate does not resolve, but is invaded by proliferating fibroblasts and capillary loops, and gradually replaced by fibrous tissue, a process known as organization. Similarly, abscess cavities after the infection is overcome are obliterated by granulation tissue growing in from their walls.

Alternatively, when peritoneal reaction has failed to localize the irritant process, a diffuse peritonitis will result, causing, if untreated, death of the patient from water and electrolyte disturbance, or resolving, with or without the formation of secondary abscesses

The factors which determine whether the fibrin matting is removed, or whether it is transformed into fibrous adhesions are not clearly identifiable. Every surgeon must have had occasion to marvel at the pristine integrity of a re-opened abdomen, last seen in the state of acute peritonitis; equally remarkable are the multiple tough adhesions which have apparently followed an aseptic procedure.

The experimental use of cortisone and ACTH certainly delays adhesion formation, together with the general fibroblastic healing process, but opinions are divided as to the long-term results from the use of these drugs.

Response of Peritoneum to Injury. The prodigious regenerative powers of the peritoneum are well known. Few surgeons now attempt the re-peritonealization of large raw surfaces in the abdomen since these become covered with primitive peritoneum within 48 hours, and intestinal adhesion to the raw surface is no more frequent than to the catgut stitches which are used for peritoneal apposition. The repair of peritoneal defects is probably by marginal ingrowth rather than cellular migration.

GENERAL PATHOLOGICAL EFFECTS OF PERITONITIS. The onset of acute diffuse peritonitis is accompanied by shock; the diminished venous return giving rise to an increased pulse rate and to fall of the blood pressure Recovery from this phase is usual, but is soon succeeded by deterioration caused by water and electrolyte loss from vomiting, [illegible] swiftly and it [illegible] hours of the

inception of the infection. Losses from vomiting are principally those of water and chloride, approximately equivalent to isotonic saline. The constitution of the peritoneal exudate approximates to that of plasma, while the fluid exuded into the intestinal lumen contains water and electrolytes, including large amounts of potassium.

Toxæmia. Absorption of toxic products from the peritoneum has been advocated as a prime factor in the rapid decline of patients suffering from peritonitis. Experimental work would indicate that absorption is inhibited when peritonitis becomes established; it cannot be denied, however, that the evacuation of large quantities of pus from the abdomen often leads to an instantaneous improvement in the condition of the patient.

The changes occurring in the constitution of the blood consist of hæmo-concentration, loss of electrolytes, and leucocytosis. Hæmo-concentration is dependent on the extent of the peritonitis and the rate of onset. Sodium and chloride loss occur early. Derangement of potassium balance may not be established by blood estimations until 48 hours have elapsed.

The leucocyte response in peritonitis varies with the severity of the infection and the age and fitness of the patient. Whereas a healthy young adult will usually show a sharp leucocytosis of 20,000 white cells or more within 24 hours of the onset of a general peritonitis, in old or debilitated patients this response may be delayed or may never have time in which to develop. The exhibition of antibiotics would also appear to be associated with lower white cell counts than might be normally expected.

CLASSIFICATION

Two main types of peritonitis have been distinguished.

(1) *Primary peritonitis*, a rare condition in which inflammation occurs without a local pathological cause in the abdomen

(2) *Secondary peritonitis*, which results from spontaneous inflammation or perforation of the abdominal contents, or follows operative intervention.

Further descriptive subdivision into acute, subacute, chronic, diffuse, spreading, localized, chemical, irritative, and terminal may be employed. In practice, acute secondary peritonitis is the most frequent and important state with which the surgeon is concerned.

ACUTE SECONDARY PERITONITIS

Secondary peritonitis may be divided into three classes:

(1) Secondary peritonitis, resulting from autogenous inflammation or perforation of the abdominal contents.

(2) Post-operative peritonitis.

(3) Peritonitis following accidental wounding.

Acute Peritonitis is most commonly secondary to acute appendicitis Other acute abdominal inflammations of the Fallopian tubes, the gall bladder, and the colon are approximately equalled in their incidence by obstruction, strangulation, and perforation of the gut, and in some localities by perforation of gastric and duodenal ulcers. Acute pancreatitis is comparatively rare, but has been diagnosed more often in recent years.

Clinical Picture. The infinite variation in symptoms and signs of acute peritonitis, from a limited pelvic tenderness on rectal examination to the classical and oft described terminal phase of the Hippocratic facies, gross distension, and "fæcal" vomiting, renders

a definitive description of peritonitis potentially fallacious. Four main clinical stages may, however, be arbitrarily distinguished.

LOCAL PERITONITIS (EARLY). Continuous pain is felt in the abdominal wall over the inflamed area. Reference of pain may occur: as from the gall bladder to the tip of the shoulder, or in retro-peritoneal inflammations, into the back. The quality of the pain is described as burning or stabbing. Vomiting does not usually make its appearance until the pain is well established unless the peritonitis is secondary to a gut obstruction. There is little constitutional disturbance at this time and many patients may not be inactivated by their symptoms. Examination will show diminished movement of the affected abdominal segment, guarding and, often, hyperæsthesia, progressing to true rigidity. Bowel sounds are frequently, but not invariably, inhibited in the vicinity, and may be normal in other quadrants of the abdomen. The sign of "rebound tenderness" has been described as invaluable in the diagnosis of early peritonitis, but the author is not convinced of its value.

LOCAL PERITONITIS (LATE). If a peritoneal response sufficient to circumscribe the local lesion takes place, a tender mass will result, with the remainder of the abdomen apparently normal.

ADVANCING PERITONITIS. Spread of the peritonitic process causes shock and prostration The pain becomes generalized and the vomiting more frequent. The patient is pale and perspires. He lies still in bed and may obtain comfort by drawing up the knees, or lying on the affected side. The pulse rate is increased, the temperature sub-normal, or rising, and respiration is shallow and thoracic. When the peritoneal effusion is highly irritant (e.g. gastric contents), the abdomen is characteristically indrawn, rigid, and universally tender, the gut motility being reduced. In peritonitis due to fæcal flooding, however, the signs are less marked Following upon the rapid advance of peritonitis and the resulting shock, a period of apparent recovery may ensue, in which the pulse and temperature steady and the pain and local signs diminish. This is followed by *established peritonitis* When peritonitis is established and extends throughout the major part of the abdomen, the general effects upon the patient predominate over the local symptoms and signs.

The picture at this time is of paralytic ileus, dehydration, and electrolyte loss. The features are sunken and grey in colour, though the apparent "brightness" of the patient may be remarked upon by the unwary. The tongue is furred and the skin may be pinched up with ease The pulse is rapid and weak, the temperature sub-normal, and the respiratory rate much increased. Abdominal distension is by now apparent. Rigidity and tenderness are decreased and silence prevails on auscultation.

The Diagnosis. John Hilton remarked in discoursing on peritonitis that "whether in medicine or surgery, nine-tenths of successful practice depends upon accurate diagnosis" "(Rest and Pain)."

The early recognition of the presence of peritonitis is of paramount importance. It is also far more easy to identify the probable source of the infection when the patient is seen before the inflammation has become general.

In the early diagnosis of peritonitis there is little to be gained by accessory aids. All depends on the clinical sense of the examiner, and it is a synthesis of history, symptoms, clinical observation, and experience which is more likely to lead to success than reliance on specific "signs of peritonitis."

In established peritonitis, radiology, by plain erect, supine, and lateral films of the abdomen, may assist, not only in confirming the clinical diagnosis, but in indicating a perforation or obstruction as the source of the infection.

For brief remarks upon conditions causing or simulating peritonitis, the reader is referred to Chapter VIII (The Appendix), p. 311.

Treatment

The principles of treatment of advancing or established acute peritonitis consist in

(1) The Treatment of shock.
(2) The Treatment of paralytic ileus and dehydration.
(3) The Treatment of the infection.
(4) The Removal of the cause.

The order of precedence of these principles will be altered by the features of each individual case.

SHOCK. The profound shock which may be seen in peritonitis such as that following perforation of a gastric ulcer requires active treatment. Operative intervention without such treatment has been known to produce fatal results.

The patient is nursed flat in bed, and the pain is alleviated by the smallest dose of morphia required for this effect. The morphia may with advantage be given intravenously. No applied heat should be employed. Restoration of the circulating blood volume should be initiated by intravenous infusion. Blood is the best fluid for this purpose, but while grouping and cross-matching are in progress, a plasma "expander" such as Dextran may be substituted.

PARALYTIC ILEUS AND DEHYDRATION As a first measure a Ryle's or duodenal tube is passed by the nose into the stomach, which is aspirated, and kept empty by hourly manual aspiration, or by continuous suction with a silent electric pump. It is desirable, if a pump is used, to connect a safety valve bottle, between reservoir and pump, to prevent excessive gastric trauma or blockage of the tube by indrawn mucosa. The emptying of the stomach will stop the vomiting, relieve the distension to a small degree, and render the induction of anæsthesia less hazardous, when the time for this arrives. After the first few hours of gastric suction decision must be taken as to the advantage which a Miller-Abbott or Cantor tube might give in a particular case. These long tubes have their use in small gut obstruction and in prolonged paralytic distension, but will not be of especial help in the majority of patients with acute peritonitis.

Should grave abdominal distension still be obvious after 24 hours, or if big dilated loops are seen on plain X-rays of the abdomen, gastric suction should be continued and a Miller-Abbott tube should be started on its way. Time should not be wasted in abortive efforts to compel its passage

Since the ileus in peritonitis is caused by peritoneal inflammation, no especial drugs except small doses of morphia for the relief of pain, and antibiotics, are required Should atony of the gut persist after the peritonitis is overcome, injections of Vitamin B complex or Pantothenic Acid may be used Prostigmine and allied preparations are rarely of service except in the deflation of an atonic large bowel Aperients are contra indicated, but small 50 per cent glycerine enemata may be of service when gut movement has begun.

Ileus which fails to respond to suction and intravenous fluid replacement must raise suspicions that the infection is not being appropriately treated, that an obstructive element may also be present, or that there exists an associated deficiency of potassium.

Supervention of a small gut obstruction on ileus secondary to peritonitis is marked by colicky pain, vomiting or greatly increased aspirations, distension, and loud peristalsis. A Miller-Abbott tube should at first be passed. If the pain persists, a laparotomy must be done.

It need scarcely be remarked, in the treatment of ileus, that no fluid should be given by mouth in quantity until bowel sounds are heard, save for occasional drinks of water to aid the patient's comfort. These are immediately aspirated back, together with any swallowed air. All intestinal contents removed by aspiration must be measured and charted. Replacement will be discussed in the next paragraph.

Dehydration and Electrolyte Loss. Water and electrolytes in the peritonitic patient are not only lost by vomiting, but also by œdema of mesentery and gut, by effusion into the peritoneal cavity, and by collection within the gut lumen as ileus increases.

The estimation of fluid requirements may be helped in some cases by weighing, if the previous weight is known, but this is seldom practicable in a patient who is seriously ill. Blood volume estimations take no account of extra-cellular and intestinal loss, and clinical judgment and common sense will usually have to take the place of more complex investigations, supplemented by a full biochemical blood examination, as soon as this can be made For example, an adult patient who has had generalized peritonitis for 12 hours will not have taken any fluid during that time, so will lack at least two pints of water for his basic requirements. Losses by vomiting may well be of the order of two pints. (Equivalent in chloride content to normal saline.) It is probable that at least two to three pints of water containing electrolytes are lying unabsorbed in the gut and the peritoneum In all, therefore, approximately 6–7 pints of fluid may require to be given in order to restore balance, consisting of 3 pints of normal 0·9 per cent saline, and 3 pints of 4·3 per cent glucose in water The route must of necessity be intravenous for absorption from the stomach and small bowel will not occur, and the rectum does not absorb sufficiently rapidly (4 pints in 24 hours) to be of real use. An intravenous drip is therefore set up, using a No. 1 or 12 short bevel needle into a distal vein in the forearm or hand.

A plan of administration of quantity, rate, and composition of intravenous fluids should be prepared, and incorporated into the patient's fluid balance chart. In a patient severely depleted administration should be begun at a rapid drip rate of 1 pint per half hour, and continued until the patient appears clinically refreshed and is passing a good volume of urine. A watch is maintained on pulse, blood pressure, neck veins, and pulmonary bases for any sign of cardiac embarrassment, but this is seldom seen provided that excessive infusion of sodium chloride is avoided. Salt and water lack will only be manifest clinically when $\frac{1}{15}$–$\frac{1}{12}$ of total body weight has been lost. The symptoms are those of thirst and lassitude. The signs include a furred dry tongue, sunken features, rapid pulse, and lowered blood pressure, and loss of skin firmness and elasticity. The urine volume is scanty and of high specific gravity. Salt deficiency can only be very roughly estimated by the rule of thumb calculations outlined above. These may be

supplemented by testing the urine for chloride (Fantus test) advocated by Marriott (1947).

Potassium lack may not occur with peritonitis until 48 hours have elapsed unless antecedent vomiting, diarrhœa, or anorexia have contributed to disturb the patient's previous normal balances. When maintenance of intravenous feeding for more than two days is necessary, it is advisable to add potassium to the routine daily requirements provided that renal function is not impaired and there is a good urine output.

Potassium chloride 3 gm. per litre (40 milli equivalents per litre) made up in isotonic solution with 4·3 per cent glucose, is a safe fluid for administration. One litre of this may be given daily for prophylaxis of potassium deficiency. An established deficiency may require up to 2 litres daily *

THE TREATMENT OF INTRA-PERITONEAL INFECTION. In the absence of shock, or following its treatment, the peritonitic patient should be nursed in a comfortable semi-sitting position. Attempts to achieve and maintain the Fowler position are disturbing to the patient, the position, in addition to its possible promotion of venous thrombosis, gives rise to impairment of respiration when the abdomen is distended, and would not seem to have the definite benefit of localizing fluid in the pelvis and avoiding subphrenic abscesses.

Small doses of morphia will be agreed by the majority of surgeons to be of apparent efficacy in peritonitis, pethidine being a possible alternative.

In acute peritonitis, antibiotic treatment should be started as soon as possible. This will of necessity be empirical until a specimen of intraperitoneal pus is obtained by operation, or, better still, until a portion of the causative pathological lesion can be cultured.

The requirements for the agent to be used are that it shall be effective against all organisms normally found in peritonitis, that it shall be able to be given parenterally and that it should be safe to use in patients whose urine output is poor.

At the time of writing, crystalline penicillin, 500,000 units combined with streptomycin gm. ½, 8 hourly, form a synergistic combination which is generally adequate. The *intravenous forms of aureomycin 500 mgm and 250 mgm. 8 hourly* and terramycin 500 mgm and 250 mgm. 8 hourly are slightly more efficacious, and should be reserved as a second line of defence unless the patient's immediate condition gives cause for alarm

The pros and cons of antibiotics have been extensively discussed in recent years. There can be no doubt that their use is justifiable in the potentially lethal condition of peritonitis, and that the dangers are a matter of secondary importance. The chief disadvantages attendant on antibiotic treatment are that:

(1) Premature administration may mask the causes of the infection before a diagnosis has been made.

(2) The course of the infection may be so modified as to add to the difficulties of treatment As an example may be adduced the formation of semi-sterile pelvic abscesses with thick walls which refuse to point into the rectum

* (For further details of potassium deficiency and its correction, together with theoretical and practical problems of fluid balance, the reader is referred to *Fluid Balance in Surgical Practice*, L. P. Le Quesne, 1954, Lloyd-Luke, London, to whom I am indebted for some of the information contained on this page.

(3) The development of resistant strains of organisms, or overgrowth of staphylococcus albus ("staphylococcal entero-colitis") pyocyaneus, proteus, and monilia may be favoured by prolonged antibiotic treatment. This risk may be minimized by restricting a course of treatment to an arbitrary five-day period; large doses of Vitamin B complex and youghourt have also been advocated by recent writers.

(4) Allergic reactions to chemotherapeutic agents may add to the severity of the patient's condition. Although these do not usually appear till some days after the administration has started, their not infrequent occurrence constitutes a minor argument against intraperitoneal or local use.

In the event of peritonitis not responding to antibiotics, it may be that the predominant organism is not sensitive to the drug in use, and that a change of agent is indicated. Alternatively, there may be a continuing source of infection which must be dealt with surgically, or resistant strains of organisms may have developed. In this last case, further chemotherapy will only make the condition of the patient worse, and measures must be started to improve the antibody response of the individual. The best means of accomplishing this is undoubtedly by the transfusion of one or more pints of fresh blood, administered as soon as possible after withdrawal from the donor.

Other agents exhibited in the treatment of peritonitis have included in the past anti-gas-gangrene serum, and anti-tetanic serum. Their use has now been largely abandoned.

Recent reports from America tell of the remarkable clinical improvement in peritonitic patients brought about by the simultaneous use of antibiotics and ACTH. This form of treatment is not yet fully appraised.

Removal of the Cause. The majority of patients suffering from acute peritonitis require operation for the removal of the causative pathological lesion, unless a primary peritonitis or a peritonitis secondary to Fallopian tube infection, or to pancreatitis, can be confidently diagnosed. The decision as to the time of the operation is of great importance.

A patient suffering from early local peritonitis secondary to appendicitis may be operated on forthwith without any preliminary treatment for shock, dehydration, or infection.

Conversely, a patient in the late stages of general peritonitis will require several hours of preparation before he will be fit for surgery, and a complete reassessment of the case will then be necessary to decide if surgery is still indicated

(The details of operations which may come to be undertaken for conditions causing or associated with peritonitis are outside the scope of this chapter, and for these the reader is referred to the accompanying chapters in this textbook.)

It is relevant, however, to consider the merits and demerits of drainage in peritonitis. These have been briefly discussed relative to appendicular peritonitis. Drainage of the peritoneal cavity for peritonitis as a sole procedure is not now practised. Many surgeons do not insert a drain following operation under any circumstances, in the belief that drainage is at best ineffective, valuable antibodies and leucocytes may be lost to the peritoneum, and that a drainage tube may give rise to gut obstruction, perforation, or residual abscesses.

In the opinion of the author there are still certain situations in which the local insertion of a slip of soft corrugated rubber, or a Penrose drain may fail to do harm and may even do good. These may be listed as follows:

supplemented by testing the urine for chloride (Fantus test) advocated by Marriott (1947).

Potassium lack may not occur with peritonitis until 48 hours have elapsed unless antecedent vomiting, diarrhœa, or anorexia have contributed to disturb the patient's previous normal balances. When maintenance of intravenous feeding for more than two days is necessary, it is advisable to add potassium to the routine daily requirements provided that renal function is not impaired and there is a good urine output.

Potassium chloride 3 gm. per litre (40 milli equivalents per litre) made up in isotonic solution with 4·3 per cent glucose, is a safe fluid for administration. One litre of this may be given daily for prophylaxis of potassium deficiency. An established deficiency may require up to 2 litres daily.*

THE TREATMENT OF INTRA-PERITONEAL INFECTION. In the absence of shock, or following its treatment, the peritonitic patient should be nursed in a comfortable semi-sitting position. Attempts to achieve and maintain the Fowler position are disturbing to the patient; the position, in addition to its possible promotion of venous thrombosis, gives rise to impairment of respiration when the abdomen is distended, and would not seem to have the definite benefit of localizing fluid in the pelvis and avoiding subphrenic abscesses.

Small doses of morphia will be agreed by the majority of surgeons to be of apparent efficacy in peritonitis, pethidine being a possible alternative.

In acute peritonitis, antibiotic treatment should be started as soon as possible. This will of necessity be empirical until a specimen of intraperitoneal pus is obtained by operation, or, better still, until a portion of the causative pathological lesion can be cultured.

The requirements for the agent to be used are that it shall be effective against all organisms normally found in peritonitis, that it shall be able to be given parenterally and that it should be safe to use in patients whose urine output is poor.

At the time of writing, crystalline penicillin, 500,000 units combined with streptomycin gm $\frac{1}{2}$, 8 hourly, form a synergistic combination which is generally adequate. The intravenous forms of aureomycin 500 mgm. and 250 mgm. 8 hourly and terramycin 500 mgm. and 250 mgm. 8 hourly are slightly more efficacious, and should be reserved as a second line of defence unless the patient's immediate condition gives cause for alarm

The pros and cons of antibiotics have been extensively discussed in recent years. There can be no doubt that their use is justifiable in the potentially lethal condition of peritonitis, and that the dangers are a matter of secondary importance. The chief disadvantages attendant on antibiotic treatment are that:

(1) Premature administration may mask the causes of the infection before a diagnosis has been made.

(2) The course of the infection may be so modified as to add to the difficulties of treatment. As an example may be adduced the formation of semi-sterile pelvic abscesses with thick walls which refuse to point into the rectum.

* (For further details of potassium deficiency and its correction, together with theoretical and practical problems of fluid balance, the reader is referred to *Fluid Balance in Surgical Practice*, L. P. Le Quesne, 1954, Lloyd-Luke, London, to whom I am indebted for some of the information contained on this page.

that some underlying and unrecognized pathology is associated with the mass. The few cases which do not resolve should be subjected to an exploratory drainage operation.

SUBPHRENIC ABSCESS. Barnard, in his classical paper on subphrenic abscess, accurately described six potential subphrenic spaces in which infected collections of fluid may be found. These are four intraperitoneal, and two extraperitoneal spaces:

(1) The right anterior intraperitoneal space between liver and diaphragm, bounded posteriorly by the right triangular ligament and medially by the falciform ligament.

(2) The right posterior intraperitoneal space (Morison's pouch) between the posterior diaphragm and kidney, and the posterior surface of the liver. Its anterior wall is the right triangular ligament and the paracolic gutter lies lateral to it.

(3) The left anterior intraperitoneal space lies to the left of the falciform ligament and is again between liver and diaphragm, the posterior boundary being the left triangular ligament.

(4) The left posterior intraperitoneal space constitutes the upward extension of the lesser sac between liver and diaphragm, limited on its antero-superior border by the left triangular ligament.

(5) The right extra-peritoneal space occupies the bare area of the liver.

(6) The left extraperitoneal space consists of the extraperitoneal area above the left kidney, and between it and the diaphragm.

Subphrenic abscess is now a rare condition in British hospitals, the incidence being of the approximate order of 0·05 per cent of general surgical patients. It is probable that many incipient subphrenic infections are now aborted at an early stage.

An abscess may occur as a complication of a perforative peritonitis of stomach, duodenum, gall bladder, or appendix; following an operation for peritonitis, or as the result of a post-operative peritonitis. More rarely, a subphrenic abscess is seen secondary to retroperitoneal or chest infection, or as a terminal event in a debilitated patient.

The spaces most commonly infected are the right posterior intraperitoneal space, and the right anterior intraperitoneal space.

Two main clinical varieties are seen. The first and more unusual is of acute onset within a short time of the primary peritoneal infection. The patient complains of pain in the right chest and hypochondrium, increased on respiration, the rate of which is itself increased Profuse perspiration and persistent hiccoughing add to his discomfort

Examination will show a high swinging temperature and a rapid pulse in a patient obviously toxæmic. Poor respiratory movement of the lower right chest may be observed. The liver edge is depressed and tender. Tenderness may also be found below the costal margin in the anterior axillary line, or below the twelfth rib. Pressure on the cervical part of the phrenic nerve is stated to produce reflex pain in the upper abdomen. Percussion usually demonstrates an increased dullness in the lower chest which may either be due to diaphragmatic irritation, or to basal lobar collapse and pleural effusion. The classical alternating zones of dullness and resonance are seldom seen.

The common clinical presentation of subphrenic suppuration is, however, far less well defined, sometimes even leading to the discharge of a patient from hospital with silent subphrenic infection well established. Failure of a post-operative temperature chart [illegible]

(1) A gross excess of intraperitoneal fluid (e.g. peritonitis supervening on ascites).
(2) Persistent oozing from a raw capillary bed.
(3) Macroscopic fæcal or foreign body soiling.
(4) Dubious viability of inflamed bowel or possible leakage from a sutured viscus.
(5) Abscess cavities which cannot be completely removed.
(6) Retro-peritoneal inflammation.

Residual Abscesses. Incomplete resolution of a general peritonitis will give rise to the formation of localized abscesses within the peritoneal cavity. In order of frequency, these are found in the pelvis, the flanks, multiple throughout the abdomen, or in the subphrenic regions.

An abscess following peritonitis may not declare itself readily, especially when antibiotics have been used. Its presence may be suspected when the pulse and temperature fail to settle to normal values, and the patient himself fails to show a progressive clinical improvement. Persistent abdominal pain, pain on deep respiration, or discomfort on passage of flatus or fæces are symptoms which may indicate the site of a localization of pus, confirmation being obtained by clinical examination, by serial white cell counts, and in subphrenic collections, by radiographic examination.

A Pelvic Abscess may present definite localizing symptoms of pain, pain on defæcation, diarrhœa, urinary frequency, or colic of involved small bowel. Nearly half the cases seen by the author have been without marked symptoms, possibly due to partial sterilization by antibiotics, and have frequently been discovered by the routine rectal examination which must always be performed on patients convalescing from peritonitis. The characteristic finding is of a tense, tender swelling in the recto-vesical or recto-uterine pouch which bulges into the rectum The mucosa will not be attached to this swelling in its early stages. The treatment is initially conservative. If the localization is deemed an early one, antibiotics may be used in an effort to secure resolution, but should be discontinued if no change occurs within three days If the abscess be well formed in spite of antibiotics, these should be stopped.

The surgeon's object is to allow spontaneous discharge to take place into the rectum without operation, and time alone will achieve this in the majority of cases, without assistance by hot douching or diathermy, which carry certain minor risks. During this waiting period therapeutic endeavour should be directed to improving the general condition of the patient. When the abscess does not diminish or point, operation is indicated, and surgical drainage may be effected under general anæsthesia by inserting a finger into the most prominent part of the boggy mucosa overlying the swelling. Should the abscess have a tough wall, a probe-pointed director and sinus forceps must be used, the abscess being entered by Hilton's method. No tube need be inserted provided that the drainage ostium is kept open by periodic digital examination. A persistent pelvic abscess which fails to point into the rectum or vagina will require laparotomy. The coils of small gut adherent to it must be gently separated and the greater part of the abscess wall removed Small bowel may then be allowed to fall into the dead space, and extra peritoneal drainage should be provided

Flank Abscesses give rise to pain in the abdominal wall over the collection and may usually be felt with ease The majority of these collections resolve and do not require drainage. Failure of resolution must raise suspicions in the surgeon's mind

advocate an approach through the bed of the eleventh rib with careful blunt upward dissection of the pleura.

The anterior extraperitoneal approach is made either by finger dissection between the peritoneum and the posterior rectus sheath, or by detaching the muscles of the abdominal wall from the costal margin and passing the fingers up superficial to the peritoneum.

The chief complications of subphrenic abscess are those of perforation into the chest or peritoneal cavities. The occasional simultaneous presentation of an empyema in conjunction with a subphrenic abscess forms a difficult problem for the surgeon. The better course in treatment is to aspirate and sterilize the empyema with antibiotics and then to drain the subphrenic abscess, provided that the two do not communicate directly. Under these circumstances double drainage both of chest and abscess may have to be employed.

Post-Operative Peritonitis

This variety of peritonitis may be caused by:

(*a*) A continuation of a pre-operative infection.

(*b*) Introduction of bacteria.

(*c*) Liberation of infective material during operation.

(*d*) Leakage, perforation, hæmorrhage, or obstruction in the post-operative period.

(*e*) Fortuitous development of a pathological process not associated with the operation. (e g. Acute appendicitis, perforated peptic ulcer, mesenteric embolism)

There are two distinct types of post-operative peritonitis. In the first a sudden onset of pain, collapse of the patient, and abdominal rigidity is a clear indication of disaster.

Far more frequently, however, the peritonitis is of insidious onset, without obvious pain or rigidity. Alterations in pulse rate and temperature may not be conspicuous in the post-operative period and the only positive signs may be those of a persistent paralytic ileus, with abdominal tenderness, distension, and silence

Treatment of post-operative peritonitis is first indicated, as in spontaneous peritonitis, for shock, dehydration and fluid imbalance, and infection. Success depends upon accurate diagnosis of the cause. If it is considered that the ætiology of the infection lies in categories (*a*), (*b*), or (*c*), conservative treatment is the right policy and should be vigorously pursued. On the other hand, peritonitis due to suspected pathology in groups (*d*) and (*e*) must be treated by laparotomy as soon as the general condition of the patient renders this safe

Re-operation has, in the past, been considered hazardous, but is withstood well by the majority of patients and may prove a decisive factor in recovery. The procedure to be followed may entail such measures as relief of obstruction, catheter drainage of a duodenal stump, or fæcal diversion by a proximal colostomy No repair of a dehiscent anastomosis should be attempted without proximal diversion, and drainage to the origin of the peritonitis should be employed in all cases

Traumatic Peritonitis. Peritonitis may follow any abdominal injury, even of trivial character Concerning the diagnosis and treatment of an abdominal wound, there are no alternatives. Following the necessary pre-operative preparation, exploration of the wound must be urgently made, and a full examination of the peritoneal cavity and contents must be done if the peritoneum has been transgressed.

leg veins, and abdomen. Failure to find a source must concentrate suspicion upon the subphrenic areas.

Conditions which have some clinical features in common with subphrenic abscess are infected basal collapse of the lung, pleural effusion and empyema, and liver abscess. Diagnostic endeavour must be applied to the exclusion of these.

Accessory aids to diagnosis include the repetition of total and differential white cell counts. Persistent polymorphonuclear leucocytosis confirms the persistence of infection.

An X-ray examination of chest and sub-diaphragmatic areas should be made, and if necessary repeated till inimpeachable films and views are obtained. Some possible positive findings are listed below:

(1) Effusion into the costo-phrenic angle with basal collapse of the lung. These appearances may not necessarily be secondary to a subphrenic abscess.

(2) Impaired mobility and elevation of the diaphragm on screening should be checked by comparison with the pre-operative X-ray.

(3) Sub-diaphragmatic gas may be observed. This may have persisted since an operation, or may be, in reality, intragastric.

(4) A fluid level seen between diaphragm and liver is diagnostic of a subphrenic abscess

In a difficult case, the radiologist may be given additional help by aspirating an associated pleural effusion or empyema, emptying the stomach of gas by a nasal tube, or, if the manœuvre is considered safe by the surgeon, by inducing a small pneumoperitoneum. Exploratory needling of the subphrenic spaces is avoided until drainage is imminent.

Treatment. An established sub-phrenic abscess, accurately located by X-ray, should be drained. If an early infection is suspected, or if there are doubts as to the site, expectant treatment should be instituted, with the intention of aiding resolution, or producing a well localized lesion. The former objection to this waiting policy, that the patient may be gravely weakened by pyrexia and toxæmia, is now discountenanced, since these symptoms may if necessary be controlled by antibiotics.

The patient is nursed in a semi-sitting position and full respiration is encouraged Particular attention is paid to maintenance of his nutrition and physical condition by high caloric and protein feeding (by tube if necessary), added vitamins, and by fresh blood transfusion should decline of the hæmoglobin level occur. Antibiotic treatment should be started, or if already in progress, a more potent antibiotic of wider application must be substituted.

The clinical appearance of the patient, daily white cell counts and X-ray examinations will indicate the success or failure of this treatment Failure to improve, or deterioration, show the need for drainage, which is immediately preceded by extra-pleural exploration with a long needle.

Drainage. A general anæsthetic is desirable The usual routes of exploration are on the right side, posterior sub-pleural, and anterior extraperitoneal, and on the left side posterior sub-pleural. Extra-pleural or sub-pleural drainage is carried out by excising the twelfth rib and making a horizontal incision through its bed. Two fingers are then passed up in the areolar tissue between liver and diaphragm till pus is encountered, and a drainage tube is inserted. Those experienced in thoracic surgery

The clinical presentation is that of an acute peritonitis without the same severity of abdominal symptoms and signs as might be expected. Immediate diagnosis is not often established except by laparotomy and by the examination of material removed by biopsy. Although operation is undesirable in a patient with generalized tuberculosis, the definite and rapid information which may be gained will usually outweigh this consideration, as it allows of active anti-tuberculous treatment to be instituted as soon as possible. In addition to anti-tuberculous drugs, it is desirable to use antibiotics of more general application if secondary organisms are present.

Chronic Tuberculous Peritonitis occurs in ascitic and adhesive forms, and in combinations of these two types. The gross peritoneal effusion is usually seen in children and young adults in whom resistance is poor. Older patients are prone to develop numerous adhesions and strictures.

Ascitic Peritonitis presents an increasing abdominal distension accompanied by fever. Alteration of bowel habit is common. Examination discloses a shiny distended abdomen on which the superficial veins are abnormally noticeable. Shifting dullness is found in the flanks, and the dome of the belly is tympanitic.

Adhesive Peritonitis is slower in onset and produces less constitutional disturbance. Colicky pain is a prominent symptom, sometimes associated with vomiting, and visible peristalsis may be seen. Thickened coils of gut are palpable in many cases, and auscultatory examination may reveal unusually loud borborygmi.

The diagnosis is aided by a familial or previous history of tuberculosis, positive clinical or X-ray chest findings, a positive Mantoux test, plain X-rays of the abdomen, which may show distended loops of gut, fluid and calcified glands, and, finally, by fæcal examination and gastric aspiration for tubercle bacilli.

If doubt still exists, a minor laparotomy and removal of omentum or other suspicious material for biopsy is the best method of establishing the diagnosis quickly. Peritoneoscopy has a place in direct diagnosis, but does not readily admit of biopsy.

The treatment of tuberculous peritonitis is primarily medical and best conducted in a sanitorium. Rest, and a low residue high protein and caloric diet are the foundations. Chemotherapeutic agents have proved effective in assisting the resolution of the disease, though relapse following their discontinuance is sometimes seen. Streptomycin grams 1 daily by injection, para-amino salicylic acid, and isonicotinic hydrazide, should be administered concurrently for at least a month The frequent secondary anæmia will require iron.

Deep X-ray therapy given in small doses of 100 R weekly for six or eight weeks has shown good results in cases in which its uses was deemed justifiable, and it is considered by some that small doses of calciferol may also be beneficial.

Surgery is only indicated in tuberculous peritonitis to confirm diagnosis, or to alleviate a gut obstruction which may be due to stricture or adhesions, if this fails to respond to the use of the Miller-Abbott tube.

It has been stated by eminent authorities, including Dr. Charles Lakin, that a therapeutic laparotomy with evacuation of ascitic fluid has in the past often brought about a striking improvement in patients when conservative measures were ineffective (the author has insufficient personal experience of this procedure).

Chylous Peritonitis. The presence of chyle in the peritoneum is occasionally noted, usually in association with a chronic inflammatory state which has caused fibrosis

Closed abdominal injury, however, presents a more arduous problem in diagnosis and in treatment. All surgeons are aware that rupture of the cæcum, duodeno-jejunal flexure, liver or spleen can be caused by minor accidents involving the abdomen, and that these patients may feel fit, appear fit, and have no signs of an intra-abdominal lesion. A radiological examination, which fails to show gas under the diaphragm may also be misleading.

It would seem desirable, owing to the present trend of legal opinion in Great Britain, that the majority of patients giving an history of abdominal injury, however trivial, should be admitted to hospital for prolonged observation.

PRIMARY PERITONITIS AND PARTICULAR VARIETIES OF PERITONITIS

In the primary category it is customary to place infections from the pneumococcus, streptococcus, gonococcus, acute tuberculous peritonitis, and mesenteric adenitis. It is doubtful, however, whether any of these infections, with the exception of mesenteric adenitis, can arise spontaneously in the peritoneum without a primary focus of bacterial invasion elsewhere in the body. Rarely the primary infection is distant, and the spread to the peritoneum by the bloodstream. Almost invariably, these special varieties of peritonitis occur secondarily to infection of the gut or Fallopian tubes.

PNEUMOCOCCAL PERITONITIS is seen in two clinical forms.

Young female children may incur the infection from pneumococci present in the vagina which ascend the Fallopian tubes. It is manifested in adults in association with a lobar pneumonia, or pneumococcal meningitis.

For description of the clinical features of this uncommon and almost extinct disease, recourse to other writers gives the clinical story of sudden onset of abdominal pain, high fever, herpes labialis, cyanosis and rapid respiration, a pinched countenance with working of the alæ nasi, and a rigid abdomen with bilateral tenderness. It has been stated that a fairly confident diagnosis of pneumococcal peritonitis to the exclusion of appendicitis, can be made, and that conservative treatment is desirable.

STREPTOCOCCAL PERITONITIS has been recorded in association with streptococcal septicæmia, due to such conditions as respiratory infection, scarlet fever, and puerpural sepsis. It may also follow upon operations and possesses no distinguishing clinical feature save for the characteristic thin serosanguineous peritoneal effusion.

GONOCOCCAL PERITONITIS arises invariably from a Fallopian tube infection. It has been described consequent on vulvitis in children, but is characteristically a disease of adult women Secondary infection with other organisms is frequent, and a culture of the gonococcus may be difficult to obtain. Dense pelvic adhesions are found at operation The diagnosis is sometimes indicated by the diathesis and deportment of the patient. Assistance may be gained by the examination of a cervical smear under the microscope. In doubtful circumstances, laparotomy is indicated to exclude an acute appendicitis.

Tuberculous Peritonitis. Three kinds of clinical tuberculous infection may be recognized. All are secondary to primary infection from the gut, Fallopian tubes or lungs.

Acute Tuberculous Peritonitis is classically associated with miliary tuberculosis, but may also be caused by perforation of the bowel in which tuberculosis is already present. One such case was seen consequent upon the perforation of an acutely inflamed appendix in which there was already an active tuberculous focus

treated by rapid and adequate blood transfusion. If the expected improvement in condition does not materialize, or if abdominal signs are manifest, laparotomy is indicated. (One of the most reliable symptoms of free blood in the abdomen of a conscious patient is that of shoulder-tip pain in recumbency.)

Spontaneous intra-abdominal hæmorrhage is most often caused by rupture of an ectopic gestation. Bleeding from a Graafian follicle in the mid-menstrual phase may give rise to a picture of lower peritoneal irritation indistinguishable from appendicular peritonitis. Spontaneous rupture of the spleen and leakage of aneurysms are comparative rarities. In all cases of suspected abdominal hæmorrhage exploration is wiser than speculation.

MYXOMATOUS PERITONITIS. "Pseudo-myxoma Peritonei."

Myxomatous or jelly-like material within the peritoneum results from rupture of a mucocœle of the appendix, or of a pseudo-mucinous cystadenoma of the ovary. It may be found to a minor extent when a mucus secreting intestinal carcinoma has disseminated across the peritoneal cavity. The majority of pseudo-myxomata arising from the appendix are secondary to papillary carcinoma in a mucocœle.

Operative treatment consists in evacuating the peritoneal cavity and removing the primary lesion. Biopsy material should be obtained from the peritoneal surfaces in order to indicate the prognosis, which does not seem to be influenced by measures such as swabbing the peritoneum with perchloride of mercury, or by barrage irradiation.

TALC GRANULOMA. The propensities of talc crystals for provoking foreign body reactions within the peritoneum have been recognized in recent years, and many surgeons now insist upon soluble non-reactive powders for use on their gloves. Provided that the gloves are carefully washed before the hand is introduced into the abdomen, and that the lotion bowl is subsequently changed, the risks of inducing adhesions are probably not large. The potential sterility which might result in a young female patient from talc introduced during appendicectomy must however, be borne in mind.

MECONIUM PERITONITIS. A low-grade peritonitis in new-born infants is recorded as the result of meconium in the peritoneal cavity. This may be the sequel to congenital gut obstruction, but can occur by spontaneous intestinal perforation. (The subject has been well reviewed by Tempest (1952), and by I. Forshall, E. G. Hall, and P. P. Rickham (1952)

References

Aird, I. (1949) 554. A Companion in Surgical Studies. *E. & S. Livingstone Ltd.*, Edinburgh.
Barnard, H. L. (1908) *Brit. Med. J.* 1, 429.
Boling, [illegible]
D[illegible]
F[illegible]
H[illegible]
[illegible], A. E. (1935) Surgical Pathology of the Peritoneum, *J. B. Lippincott*, Philadelphia.
Le Quesne, L. P. (1954) Fluid Balance in Surgical Practice, *Lloyd-Luke*, London.
Marriott, H. L. (1947) *Brit. med. J.* 1, 245, 285, 328.
Mitchell, G. A. G (1940) *Brit. J. Surg.* 28, 291.
Scheinberg, S R. and Saltzstein, H. C. (1951) *Arch. Surg.* 63, 413–420.
Stringer, P. (1955) *Arch. Middx. Hosp.* 5, 42.
Tempest, M N. (1952) *Brit. J. Surg.* 40, 28.
Thompson, M. and Buschemeyer, W. (1952) *Ann. Surg.* 135, 615.

of the thoracic duct. This subject has been reviewed by Thompson and Buschm (1952).

BILIARY PERITONITIS. Distinction must be made in this condition between a l stained peritoneal effusion associated with cirrhosis, and a primary effusion of bile i the peritoneum. Biliary peritonitis is most often due to injury, or to leakage follow operation caused by such mishaps as displacement or blockage of the T-tube. St bile, in particular after operation, may cause very little in the way of acute symptom signs, and a large quantity can accumulate in the belly without its presence being rec nized. Infected bile, on the other hand, causes a fulminating and sometimes f peritoneal reaction.

Displacement of the T-tube may be confirmed by gentle saline irrigation, and r ography after instillation of diodone. Laparotomy may be necessary for evacua of bile from the abdomen and replacement of the tube.

Spontaneous or primary biliary effusion ("biliary dew") has for long excited curiosity of the general surgeon since the condition was first described by Leriche. case appeared to be consequent upon an acute cholecystitis which had rendered the bladder permeable. Other cases have been recorded in which there was necrosis of common bile duct secondary to pancreatitis, and perforation of a crypt in the wall been adduced as an explanation. In the remainder of reports, no pathological ca has been discovered.

ACUTE NON-SPECIFIC MESENTERIC ADENITIS. Non-specific enlargement of mesenteric lymph nodes, accompanied by a serous effusion, and peritoneal hyperæn may occur as part of a generalized lymphadenoid hyperplasia due to respiratory in tion or infectious mononucleosis. It may also be seen as a response to allergy or infect of the small intestine.

The clinical picture is of a child who is smitten by intermittent severe colicky abdc inal pain and fever. Previous episodes may be recollected by the mother. Ther usually little guarding or rigidity, but marked tenderness is found in the right iliac fo where glands may on some occasions be felt.

Ian Aird, who has written from a wide experience, states that the sign of "shift tenderness" may be of value. A careful search should be made for faucial inflammat and for glands in the neck, axillæ and groins. A differential white cell count will sho mild leucocytosis with relative lymphocytic or mononuclear increase.

The chief difficulty in diagnosis of mesenteric adenitis lies in its resemblance appendicitis, and in the fact that lymphoid hyperplasia may not only have affected mesenteric glands, but also the lymphoid tissue in the appendix itself. Unless the surge confronted with such a case, in emergency, is confident of his ability to distinguish t two conditions, appendicectomy is wiser than conservative treatment. Glands remov for histological examination in this disease are usually reported as showing "nc specific reactive hyperplasia."

HÆMATOMA OF THE PERITONEUM. Blood in the peritoneal cavity may give rise marked peritoneal irritation. On the other hand, symptoms and signs are often insignificant that its presence may be a surprise to the operator.

Following closed abdominal injury, it is at first difficult to distinguish between sho and intraperitoneal hæmorrhage unless the bleeding is brisk. Furthermore, distinctic of intraperitoneal from retro-peritoneal bleeding may not be easy. The shock must

may be an isolated one but usually there is direct continuity with a lesion of the ileum. Very much less frequently the disease involves the jejunum and rectum, and some claims have been made for its discovery in the stomach and œsophagus. The length of ileum affected varies very considerably, but is on average less in the acute cases than in the chronic ones. Rather more than half the acute cases have less than 12 in. of terminal ileum involved, whereas in the chronic condition two-thirds of the cases have a foot or more. Rare cases where the whole ileum and part of the jejunum (Crohn) and the whole of the small bowel (Dalziel) were involved have been reported. One of the features of the condition is the possible presence of "skip areas" of healthy bowel between two or more lengths of diseased bowel. This occurred in 5 of the 34 (15 per cent) cases described

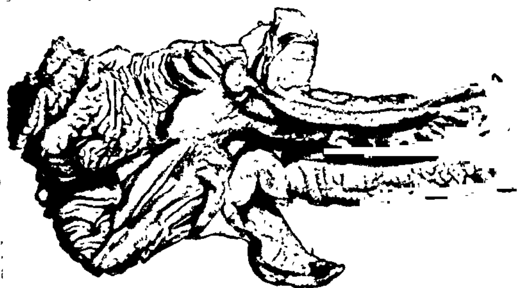


FIG 149. Regional ileitis

by Armitage and Wilson (1950). These authors at the same time, however, point out that multiplicity of lesions is a feature more characteristic of tuberculosis than "non-specific ileitis." The disease is encountered clinically in acute and chronic phases, but it must not be supposed that the order of sequence is always the same. It is probable that in most cases superadded infection to the chronic lesion leads to the acute manifestations of the condition.

Gross Appearances. Descriptions of the bowel in either phase are of well-developed pathological changes and there is no real evidence to indicate the nature of the initial lesion. It is believed by some that the primary lesion is a small ulcer on the mesenteric border of the bowel, and that this forms the portal of entry for infection. In the developed condition, which is the one seen by surgeons, the two main findings are the thickened bowel and *enlarged mesenteric lymph nodes*.

The bowel shows a fairly abrupt change from the normal to the diseased ileum. The surface of the bowel is typically reddened, and it may be covered with a fibrinous exudate. The greatly thickened wall has lost its normal suppleness, and the feel of it has been variously likened to that of a rubber hose or "an eel in a state of rigor mortis" (Dalziel). The lesion of the ileum usually ends at the ileo-cæcal valve, but in some cases extends to

CHAPTER X

CROHN'S DISEASE

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IN 1932 Crohn introduced the term "regional ileitis" to describe an inflammatory condition of the terminal ileum. He defined it as "a non-specific chronic recurrent granulomatous disease affecting mainly young adults, and characterized by a necrotizing, ulcerating inflammatory process, one in which cicatrizing elements in the long-enduring cases are an important feature." Pathological changes similar to those found in the terminal ileum have been found in other parts of the gastro-intestinal tract. Because of this attempts have been made to use a term of a more comprehensive character than "regional ileitis," and in the absence of knowledge of the exact pathology of the condition, it has been called "non-specific granuloma." The name Crohn's disease is, however, well established, and in addition to giving credit where it is due, it brings to mind a definite clinical concept of a condition which is not at all uncommon.

HISTORY

When a condition has been clearly described and its symptoms classified, cases noted in the past are recognized to be of the same nature. In 1813 Coombe and Saunders described a case in which "the lower part of the ileum as far as the colon was contracted for the space of three feet to the size of a turkey quill." This was reported as "a singular case of stricture and thickening of the ileum." In 1828 Abercrombie described the post-mortem findings in "a girl aged 13, who about a year before her death, began to be afflicted with pain of the abdomen and frequent vomiting. The caput coli three inches along the ascending colon and the lower end of the ileum to the extent of about eighteen inches, was distended, thickened in the coats, externally of a reddish colour, and internally covered by numerous well-defined ulcers, varying in size from a diameter of a split pea to that of a sixpence."

In 1913 Dalziel anticipated Crohn by describing eight cases of "chronic interstitial enteritis," which he regarded as a disease not previously described, saying also that it seemed probable that many cases must have been seen and diagnosed as tuberculous. Many other cases can now be seen to fit the picture of Crohn's disease, and from the 1920's in particular, the number of references to non-specific granulomata of the intestine increased rapidly The work of Moschowitz and Wilensky (1923) on non-specific granulomata of the intestine calls for special mention as a step towards the differentiation of these from other inflammatory conditions of the bowel. At the time of Crohn's original article (1932), it was thought the affection of the ileum represented a condition which was distinct from other non-specific granulomata elsewhere in the intestine. There is no adequate pathological basis for making this distinction.

Pathology

Site of the Disease. In more than four-fifths of cases, the terminal ileum is affected. The cæcum and colon are next most commonly involved. Occasionally a colonic lesion

a reticulosis—a term used to group a number of mystifying enlargements of lymphoid and reticular tissues. The histological appearances of lymph nodes affected by Crohn's disease and Boeck's sarcoidosis cannot be distinguished from each other, but Crohn's disease never manifests itself in lesions of the skin, lungs, bones, spleen, and the general lymph nodes throughout the body.

It is known that *lymphatic obstruction* leading to lymphœdema may be followed by ulceration and then secondary infection. A non-specific mesenteric lymphadenitis may according to this notion be responsible for the initiation of the bowel lesions of Crohn's disease (Reichert and Mathes, 1936).

The cause of ulcerative colitis is equally obscure, and in a few cases, an association of the two conditions has been noticed either simultaneously or with the ulcerative colitis following the Crohn's disease (Cattell, Lahey, *Lancet* Ed.). Emotional disturbances are common in both conditions and appear to be of ætiological significance in ulcerative colitis. In the case of Crohn's disease *psychomatic factors* are less likely and it is more probable that any emotional instability follows the onset of the disease rather than precedes it. Other factors regarded as significant by various authors have been *foreign substances* such as silica or talc, and of lipoid substances in the wall of the intestine acting as chronic irritants. Patey (1949) noted an unusual shortness of the small intestine in two of his cases.

For the present we must conclude simply that the condition is a non-specific granuloma. There seems little to be gained in making subdivisions of these granulomata according to site in the intestine or minor variations from the usual type until a firmer pathological basis is established.

Clinical Features

The disease may occur at any age but the greatest incidence is between 20–30. The condition has been described in the post-mortem findings of a new-born infant (Koop 1947). Various collected series show that the sexes are affected equally. The occasional report of more than one case in a family may possibly mean more than can be accounted for by chance.

Symptoms. The recognition of the condition often takes place during an acute attack, although symptoms have been present in four-fifths of patients for more than a year. The symptoms are those of (*a*) an ulcerative enteritis, (*b*) obstruction and in some cases (*c*) of fistulæ. In the acute phase of the condition with its associated peritoneal irritation, the patient is usually diagnosed as having acute appendicitis, and operated upon. *Pain* and tenderness in the right iliac fossa often preceded by pain of a colicky nature are combined with vomiting and a slightly raised temperature. It may be possible to feel a mass consisting of thickened bowel and mesentery. The shape of this may suggest Crohn's disease rather than an appendix mass. Pain in the chronic phase is colicky in nature and while it varies in severity, it is usually mild. It is associated with the partial obstruction caused by the condition. In between attacks of pain, it is usual for patients to have a more generalized and persistent abdominal discomfort.

Diarrhœa is present in three-quarters of the patients, and may be preceded by colicky pain, which is relieved by defæcation. The diarrhœa is never so severe as that occurring in ulcerative colitis.

It may be present as a continuing looseness of the bowel or come in periodical

the cæcum and ascending colon. The appearances in the latter are similar to those in the ileum in most cases, but from time to time it is found that only part of the circumference of the bowel is involved. The mucosa shows ulceration, particularly on the mesenteric border, and in a longitudinal direction. The normal transverse folds of mucosa disappear. The patches of mucosa remaining between åreas of ulceration give a cobblestone effect, but there is no polypus formation such as occurs in ulcerative colitis. The narrowing of the lumen of the bowel may cause obstruction, and the bowel above the lesion will in these cases be found to be dilated and to show some thickening due to hypertrophy of its muscle. Cases of complete obstruction have been described. "Skip lesions" separated by "skip areas" of healthy bowel have been alluded to.

Adhesions between the terminal ileum and loops of intestine or other intra-abdominal structures, with or without abscess formation, are common and may be followed by the formation of *fistulæ*. These *internal fistulæ* are usually to another part of the bowel, but have also been described to the bladder, ureter, vagina, and even the Fallopian tube.

External fistulæ are almost invariably due to surgical intervention in the form of a misguided appendicectomy but also less frequently as a complication of an operative attempt to cure the condition. It is usual to find the mesentery grossly thickened and containing numbers of large, pink lymph nodes of softish consistency.

Microscopic Appearances. Varying degrees of acute, subacute, and chronic inflammation are present, with an infiltration of the whole wall by polymorphonuclear, plasma, and round cells. The most noticeable feature is the presence of giant-cell systems, strikingly similar in appearance to those found in tuberculosis. These are to be seen in the bowel wall and in the mesenteric lymph nodes. Caseation does not take place. Hadfield emphasizes and regards as a specific response the early involvement of the submucous lymph follicles which enlarge and become replaced by proliferating endothelial cells, in the centre of which a Langerhans' giant cell is usually to be found. He regards the ulceration of the mucosa as secondary to the lymphatic hyperplasia and lymphœdema of the submucosa. Fibrosis takes place *pari passu* with retrogression of the earlier hyperplastic response.

Ætiology. The cause of the condition is not known. In the past its individuality has not been recognized, and most of the cases have been regarded as hyperplastic ileal or ileo-cæcal tuberculosis. This is not surprising for in addition to a tumour-like swelling due to chronic inflammatory causes, i.e. a granuloma, there are the remarkable histological resemblances in the two conditions. However, the tubercle bacillus cannot be found in the affected bowel, and the lesions in bowel and lymph nodes never caseate. It is now suggested that there is no such thing as hyperplastic tuberculosis of the intestine (Warren and Somes, 1948), and it is certainly true that in all hospitals cases indexed as tuberculosis of the intestine have practically disappeared.

Various bacteria have been suggested as being the cause of the disease, but none has been accepted They include streptococci, b. coli, and the organisms of bacillary dysentery. It is inevitable that the possibility of there being a responsible *virus* should have been suggested, but there is no evidence in support of this. The venereal condition *lymphogranuloma inguinale* not infrequently affects the rectum in addition to its more usual sites, but appears to have no connection with Crohn's disease. The Frei test is negative. There are striking resemblances histologically between Crohn's disease and *Boeck's sarcoidosis*, and for this reason the disease has been considered by some to be

Investigations

Barium meal and barium enema examinations are the most important aids to the diagnosis of the condition. Hæmoglobin estimation and examination of the stools for occult blood should also be carried out.

A barium enema is, on the whole, more valuable than the barium meal, because the terminal ileum is not obscured by other loops of small intestine containing the opaque medium. The radiological appearances show the narrowing of the lumen of the terminal ileum. This was well described by Kantor (1934), and is known as his "string sign." Apart from narrowing, an irregularity of the mucosal pattern may be detected. Fistulous tracks and skip lesions should be looked for. Cæcal and colonic lesions occurring apart from ileal disease may give appearances which cannot be distinguished from a carcinoma.

Differential Diagnosis

Appendicitis is the condition for which Crohn's disease is most likely to be mistaken. The symptoms and signs in the acute phase are very similar, but in most cases of Crohn's disease there will be a history of symptoms, particularly of abdominal discomfort with diarrhœa extending back for several months. Even if Crohn's disease is considered, it is quite likely however that misdiagnoses will occur, perhaps with sufficient doubt in some cases to make the surgeon prefer a paramedian to a grid-iron incision. It must be remembered that acute appendicitis is a very common condition, and Crohn's disease is not, and it would be wrong to risk the consequences, possibly disastrous, of delay in surgical intervention because of some uncertainty in the diagnosis. It would be equally wrong, however, to perform an appendicectomy for Crohn's disease, because of the risk of causing an external fistula.

Tuberculosis. There is some doubt whether hyperplastic tuberculosis of the bowel exists, but it would be strong prima facie evidence that an ulcerative lesion of the ileum was tuberculous if active tuberculosis of the lungs were present. Tuberculous mesenteric lymph nodes are so common that their association with an intestinal lesion cannot be regarded as conclusive that it is tuberculous. The histological examination of the bowel may not settle the question which may only be resolved in those few specimens from which the tubercle bacillus has been cultured

Carcinoma. In older patients, the differential diagnosis may be difficult. Most cases of Crohn's disease which involve the colon also affect the terminal ileum, and a careful radiological examination of this may decide the diagnosis

Actinomycosis must be considered, and a search made for the fungus, particularly in those cases where a fistula persists after appendicectomy.

Ulcerative Colitis. The inflammatory changes of ulcerative colitis may spread upwards to the ileum but the preponderance of observable changes are to be seen in the colon and rectum. Bloody stools are usual in ulcerative colitis but not in Crohn's disease in which also the diarrhœa is less severe.

Lymphosarcoma in the ileo-cæcal region may occur rarely but is not likely to be diagnosed accurately before operation

Treatment

Only a very small proportion of cases achieve spontaneous or medical cure. A few cases seen and diagnosed in an acute phase have settled down without further symptoms.

attacks The passage of *blood per rectum* is unusual but tests for occult blood are frequently positive.

Anæmia of sufficient severity to be noticeable clinically is more often present than not.

Loss of weight, energy, and *joie de vivre*, and the development of anxiety neurosis are common.

Fistula-in-ano as a presenting symptom is somewhat unexpected, but has been recorded in many cases. In this connection it is worth stressing the difficulties of the



FIG 150 Kantor's string sign in Crohn's disease of the terminal ileum

pathologist in forming an opinion of the cause of the fistula on histological grounds alone. The giant-cells present may be of the foreign body type, or part of the tissue response to a tuberculous infection, or yet again to the infective agent of Crohn's disease.

On examination of the patient's abdomen a *palpable mass* may be found in both acute and chronic phases of the condition In the former, although the mass is present, the resistance to palpation caused by the peritoneal irritation may prevent it being felt. A mass can be felt in more than half the chronic cases In one case I had under my care of a man age 52, suffering from a regional ileo-colitis, the patient had himself noticed the abdominal mass on the right side of his abdomen

After resection recurrence occurs by a similar extension or persistence of mesenteric disease from lymph nodes which have not been removed. Recurrence may occur at any interval after operation. It may sometimes be years afterwards. The symptoms tend to be vague and of the nature of general abdominal discomfort or an unhealthy awareness of the abdomen. The presence of anæmia and a loss of weight associated with this, are very suggestive and call for radiological investigation.

Crohn, B. B. (1932) *J. Amer. Med. Ass.* 99, 1323.
Coombe and Saunders (1813) *Med. Trans. Roy. Coll. Phys.* 4, 16.
Abercrombie, J. (1828) *Pathological and Practical Researches in Diseases of the Stomach, etc. Waugh and Innes,* Edinburgh.
Dalziel, T. K. (1913) *Brit. Med. J.* 2, 1068.
Moschowitz and Wilensky (1927) *Amer. J. Med. Sci.* 173, 374
Armitage and Wilson (1950) *B.J.S*
Hadfield, G. (1939) *Lancet* 2, 773.

For the majority, however, whether met in an acute phase or a chronic phase, there is the prospect of a steady spread of the disease along the bowel, or an uneasy maintenance of the status quo. Intestinal obstruction, internal fistula formation or even in rare instances perforation of the bowel may all lie in store. The treatment of choice is surgical.

The surgical procedures available are (1) ileo-transverse colostomy and (2) resection of the affected intestine (*a*) in one stage (*b*) in two stages

In 1945 Garloch and Crohn published the results of twelve years' experience of the surgical treatment of the chronic condition. The series included cases of enteritis, terminal ileitis, and combined ileo-colitis.

The figures given were:

	Number	*Deaths*	*Recurrences*
Ileo-colostomy with exclusion . .	65	0	9 (13·8%)
One-stage resection	55	9 (16·3%)	9 (19·5%)
Two-stage resection	25	2 (12%)	8 (36 3%)

It should be noted that the two-stage resection group includes failures of treatment by anastomosis alone. The difference in the number of deaths remains impressive.

Ileo-transverse Colostomy. *In order to divert the stream of intestinal contents from* healthy ileum to healthy colon, this must always be of the exclusion type in which the ileum above the lesion is transected. The distal end of the ileum is sutured and returned to the abdomen unless gross obstruction is present, when it would be safer to leave it to open on the surface of the abdominal wall as a fistula until the condition has become quiescent. A simple side-to-side anastomosis between the ileum and transverse colon is useless The advantage of ileo-transverse colostomy over resection is in the low mortality, but it is less effective as a method of cure. The diseased bowel remains in many cases unhealed, although causing milder symptoms, and in others there is a relentless extension of the disease towards and even beyond the anastomosis

Resection. This can be done as a one or two-stage procedure. Most cases are preferably done as a primary resection, and only in the presence of difficulties arising from the patient's general condition or from local conditions such as fixity of the mass, or unusual extent of the disease, should a two-stage operation be planned. There is a risk of external fistula formation after ileo-transverse colostomy which is avoided by a one-stage resection The mesentery is usually grossly thickened and contains large numbers of swollen lymph nodes Most of these will be removed at operation, but it will not be possible to do this completely

Circumstances may make simple closure of the abdominal incision advisable in those patients operated upon under a mistaken diagnosis of acute appendicitis. The surgeon must resist any temptation to remove the appendix.

Results

Whatever form of surgical treatment is adopted, there will be a disappointing number of recurrences—as many as a third or even half in some series of the cases. After anastomosis the disease may persist in the original site, and also extend along the bowel either in continuity or less commonly by a skip lesion.

(*b*) Secondary: (i) Diverticula due to disease of the bowel wall—for example, ulcer diverticulum of the duodenum.

(ii) Traction diverticula.

(*c*) Pseudo-diverticula—for example, cholecysto-duodenal fistula.

Meckel's Diverticulum

Meckel's diverticulum is the commonest of the anomalies due to the persistence of the omphalo-mesenteric or vitello-intestinal duct and occurs in about 2 per cent of people at a point from 2–4 ft. from the ileo-cæcal valve on the anti-mesenteric border of the ileum. Shaped like the little finger of a glove, it has a wide opening into the lumen of the ileum and bowel contents can readily pass in and out, unless inflammation or ulceration narrow its base. Heterotopic gastric mucosa is found in about a quarter of Meckel's diverticula and pancreatic tissue may also be present. The duct may remain patent at the umbilicus after birth, discharging small amounts of mucus and fæces. Occasionally the small bowel may invert through this fistula like the letter "Y" and cause acute intestinal obstruction. The duct may degenerate to a fibrous band attached at the umbilicus or elsewhere in the abdomen. Cysts lined by intestinal mucosa may remain situated in the line of the duct between ileum and umbilicus and ectopic intestinal mucosa may also remain at the umbilicus, leaving a small raspberry-like tumour, the so-called entero-teratoma. The majority of Meckel's diverticula do not cause any symptoms by which they may be recognized during life. They may, however, present as acute abdominal emergencies in the following manner:

Inflammation. This is not common in a Meckel's diverticulum owing to its wide neck, but if narrowing occurs, a closed loop obstruction takes place and an inflammatory process results similar to appendicitis from which indeed it is unlikely to be clinically distinguishable Perforation and general peritonitis may follow. Tuberculosis of a Meckel's diverticulum has been reported by MacDonald (1947).

Obstruction. There are many ways in which a Meckel's diverticulum may cause intestinal obstruction Persistence of a fibrous band to the umbilical cicatrix or adherence of the tip by inflammation to the abdominal wall or neighbouring structures will set the stage for rotation, volvulus or strangulation of a loop of small bowel. This may give rise to long standing chronic abdominal pain due to a partial rotation before the onset of acute strangulation. A Meckel's diverticulum may actually twist around the loops of bowel or tie itself into a knot, though for this to occur a long diverticulum with a terminal ampulla is necessary. The formation of these remarkable knots was apparently well recognized by surgeons over a hundred years ago and several cases reported during the present century are described by Walsh (1950). Obstruction may result from intussusception of the diverticulum into the ileum headed by a nodule of gastric or pancreatic tissue. The persistence of a peritoneal caul or distorted mesentery of the diverticulum caused simple obstruction of the small bowel in a case described by Curr (1950)

Hæmorrhage and Perforation. The not infrequent occurrence of ectopic gastric mucosa in a Meckel's diverticulum may result in the formation of a peptic ulcer similar to an anastomotic ulcer in the stomach. The ileal mucosa is unable to tolerate the action of gastric juice though the secretion from ectopic pancreatic tissue does not appear

CHAPTER XI

DIVERTICULOSIS OF SMALL INTESTINE

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DIVERTICULOSIS of the small intestine is an uncommon condition but one which every abdominal surgeon meets from time to time and which provides not a few of the surprises of emergency surgery. Generally uncomplicated, diverticula are discovered by X-ray examination, accidentally at operation or during post-mortem examinations, for it is unusual for these conditions to be diagnosed clinically. Nearly all of the acute abdominal emergencies can occur as complications of diverticulosis of the small bowel and it is also becoming increasingly recognized that they may give rise to a variety of more chronic symptoms. Historically, Chomel (1710) and Morgagni (1761) have been accorded priority in being the first to describe duodenal diverticula, the earlier specimen, however, contained multiple gall stones and is now generally considered to have been a dilated common bile duct. In 1812, Johann Meckel (1781–1833) recognized the diverticulum which bears his name, and he appreciated its congenital nature as distinct from acquired diverticula Jejunal diverticulosis was described by Astley Cooper in 1807 and by Osler in 1881. Treves, in a Jacksonian prize essay in 1883 referred to the production of an intestinal obstruction by diverticula In the last quarter of the century, as a result of radiological studies and more frequent laparotomy, many hundreds of further cases have been recorded with almost every possible type of complication.

Classification. As might be expected, a certain confusion exists with regard to the classification of diverticula found over so great a length of small bowel and where the ætiology is not always clear A general distinction may be made between true and false diverticula, the former possessing a wall composed of all the layers of normal intestine, and the latter, so called mucous membrane herniæ where the muscular coat is deficient in part or whole The separation of congenital and acquired diverticula is acceptable within the limits of our knowledge, though the cause of the acquired variety is not always apparent, and to these the term primary is given. Secondary acquired diverticula refer to those resulting from a known cause, such as duodenal ulcer, or where they may be produced by traction from without In searching for an understandable and workable classification of diverticula of the small bowel, there is no need to look further than that of Edwards (1948) whose contributions to this subject form the basis of most British articles on diverticulosis

(1) *Congenital Diverticula*
- (*a*) Meckel's diverticulum
- (*b*) Non-Meckelian: (i) Giant diverticula
  (ii) Cyst-like diverticula

(2) *Acquired Diverticula.*
- (*a*) Primary or hernial type.

tubulation state. Fig. 151 shows such a cyst-like diverticulum removed from a 3-year-old child who had had melæna. The cyst had a well defined muscular coat and contained ectopic gastric mucosa and a chronic peptic ulcer.

Acquired Diverticula

Primary Duodenal Diverticula. Duodenal diverticula are always of the acquired variety; they may be primary, due to herniation of the mucous membrane or secondary, to duodenal ulcer or very rarely to neoplasm or traction from without.

Primary duodenal diverticula are found in the second, third, and very rarely in the fourth parts of the duodenum. The vast majority of diverticula arise on its concave border in relation to the bile and pancreatic ducts and at the point of entry of the blood vessels. A small proportion occur on the anterior or convex surfaces, 21 out of 349



FIG. 152. Three examples of duodenal diverticula X-ray films showing fluid levels.

duodenal diverticula described by Weintraub and Tuggle (1941) being in this situation Rounded or oval in shape and often as large as golf balls, the diverticula open by wide mouths into the lumen of the duodenum. They are not infrequently multiple and may co-exist with diverticulosis of the colon The incidence of diverticula found in routine radiological examination of the upper intestinal tract will vary with the care taken in the examination, and with the size of the diverticula and the number of patients with upper abdominal symptoms referred for X-ray. About 2 per cent is probably the average finding. Diverticula of the duodenum have very rarely been recorded before the age of 30, the average age at which they are diagnosed is 53. The pathogenesis of acquired primary duodenal diverticula has given rise to considerable speculation Their age incidence suggests that some factor must have been operating for many years and their position on the concave border of the duodenum in relation to ducts and blood vessels shows that the original herniation occurred at the weak spot provided at these points. It is known to be possible for the duodenal pressure to rise to considerable heights although this may not be a normal feature for this part of the bowel but one which is due to atypical spasm from one cause or another. Once initiated there is little to prevent the further development of the diverticula provided the patient survives long enough.

to be harmful. These peptic ulcers may give rise to post-prandial pain, hæmorrhage or perforation. Symptoms generally occur during childhood and three-quarters of the recorded cases of such bleeding ulcers have occurred in the first 15 years of life and this symptom is rare after the age of 30. The presence of a Meckel's diverticulum should be suspected in children suffering from intestinal hæmorrhage when other likely causes have been excluded. The ulcer is generally situated at the base of the diverticulum or just on the adjacent ileal mucosa. When perforation occurs it is stated by Cobb (1936) to have a particularly high mortality due, no doubt, to the age of the patient and the



FIG 151 Congenital cyst-like diverticulum of the ileum

nature of the ileal contents Perforation of a Meckel's diverticulum has occurred without the presence of a peptic ulcer due to impaction of foreign bodies or food particles, MacFarlane (1948) and Ward-McQuaid (1950).

Treatment. When discovered accidentally during laparotomy for some other condition, a Meckel's diverticulum should be removed if the circumstances permit. The diverticulum is amputated across its base and care taken to avoid narrowing the ileum during the closure *It is important to examine this portion of the bowel when disease* expected in the duodenum or the appendix is not found. When acutely inflamed, it is generally possible to amputate the diverticulum at its base in the same way as performing an appendicectomy Resection of the affected loop of bowel may, however, be necessary if strangulation intussusception or ulceration of the ileum has occurred.

Congenital Non-Meckelian Diverticula. These are of great rarity. Giant diverticula of the ileum are apparently a variety of duplication of the bowel due to an error in canalization Congenital cyst-like diverticula may arise along the mesenteric border of the small or large intestine and are commonly solitary The origin of these is probably similar to that of enterogenous cysts, a sequestration of the intestinal anlage during the

Jejunal and Ileal Diverticulosis

iose in the ileum. Both types are far less common than either Meckel's diverticula or iose in the duodenum or colon with which, however, they may be associated. Their



Fig 153 Jejunal diverticulosis.

incidence is said to be greater in men than women, all age groups being affected but most cases are diagnosed between the ages of 60 and 70. Cases have, however, been recorded as young as 13 and 20. Jejunal diverticula may occur singly but more commonly are multiple and many hundreds may be present along the length of the small bowel. These are commonest in the upper part of the jejunum and they decrease in frequency downwards. Once the tendency to formation has started, the condition is probably a progressive one in which the diverticula become larger and more numerous. Their pathogenesis is similar to that of other acquired primary diverticula where a herniation of mucous membrane occurs on the mesenteric border in relation to penetrating blood vessels commencing as paired bulges which coalesce and which ultimately bulge on either side of the mesentery giving an appearance which one cannot fail to recognize. Various theories have been advanced to explain the origin of these bulges: irregular peristalsis, deficiency of the longitudinal muscles on the mesenteric border of the jejunum and arteriosclerosis of mesenteric vessels which by contraction may pull out the small initial hernia. Many hundreds of cases have been recorded in the literature with accounts of their complications, Gerster (1938), Benson, Dixon, and Waugh (1934) and Milnes Walker (1945)

Each contraction of the bowel will tend to push it further out and weaken its walls which eventually become thin and deficient in muscle coat.

Symptoms. The majority of duodenal diverticula are symptomless but when symptoms do occur they are vague and likely to be confused with those of a peptic ulcer, gall bladder disease, visceroptosis or colitis. Edwards has noted that flatulence is not uncommon. When discovered radiologically, as will almost always be the case, it is important to exclude by every possible means that the diverticulum is in fact the only lesion and that the symptoms are not due to some more common adjacent pathological condition. Small diverticula are unlikely to give rise to symptoms but these are more likely to be present when the diverticulum reaches a large size and delay in emptying, often for as long as 24 hours, occurs. Fortunately, the contents of the upper intestinal tract are both fluid and sterile and diverticulitis with such complications as perforation or hæmorrhage occurs infrequently. Ferguson (1953) describes a case of perforation of an infected duodenal diverticulum and emphasizes the difficulty in detecting the condition even at laparotomy. Œdema and exudate lying to the right of the second part of the duodenum may be a guide to the correct diagnosis. Much rarer complications of duodenal diverticula have been recorded when the pressure of the diverticulum obstructs the common bile duct giving rise to jaundice, cholangitis, and pancreatitis. It has even been known for partial obstruction of the duodenum to occur notably when the diverticulum is at the duodeno-jejunal junction.

Treatment. The decision to operate on a duodenal diverticulum must be taken with great circumspection for their removal is not devoid of risk and indeed may not cure the patient's symptoms. The dangers of operation lie in the fact that most diverticula lie retro-peritoneally, where absence of a peritoneal coat makes the closure of bowel more hazardous, and to their proximity to the bile and pancreatic ducts. Medical treatment should always be given a trial in the smaller diverticula, operation being reserved for the large and slowly emptying diverticula where no other lesion has been found. At the time of operation the diverticulum may be difficult to find or palpate for it lies in an inaccessible position and collapses very readily. The majority are known to be in the second part of the duodenum and 83 per cent occur in this situation. They should be approached by dividing the peritoneum on the lateral side of the duodenum which is mobilized medially The diverticulum will then be found as a thin walled sac in relation to the bile and pancreatic ducts It may be helpful to open the lumen of the duodenum and to place a finger in the diverticulum to facilitate dissection. After removal the duodenal wall is closed by layers of interrupted thread sutures or if the neck be narrowed, by a pursestring suture. Cattell (1951) recommends the use of the long T-tube in the common bile duct as an added precaution when resecting diverticula near this duct. In excising the diverticulum, a cuff of mucosa should be retained which may facilitate better closure. Operations such as gastro-enterostomy find no place in the treatment of duodenal diverticula.

Secondary Duodenal Diverticula

Ulcer diverticula result from healing and scarring of duodenal ulcers and not from the floor of the ulcer giving way They are seldom large and give rise to the trefoil appearance of the duodenum Their wall consists of all the normal layers of the bowel. No special treatment is indicated except that for the ulcer which has produced them

References

1712, p 37.

408.

Brit J. Surg. 34, 218.

Treves, F. (1883) Intestinal Obstruction. Jacksonian Prize Essay. p. 45.

Walker, R. Milnes (1945) The Complications of Acquired Diverticulosis of Jejunum and Ileum, *Brit. J. Surg* 32, 457.

Walsh, A. (1950) Knot in Meckel's Diverticulum Causing Acute Intestinal Obstruction, *Brit, J. Surg.* 37, 475.

Symptoms. Most of the jejunal diverticula remain symptomless and unsuspected but when complicated they may present as follows:

DYSPEPSIA. A flatulent dyspepsia commonly suggests other conditions than jejunal diverticula but when this symptom is associated with pain predominantly in the left upper quadrant of the abdomen and especially if noisy borborygmi are complained of, the possibility of this condition should be considered and it may well be demonstrated radiologically.

INFECTION. This is not common as the contents of the jejunum are not highly infective. Acute diverticulitis may, however, occur with or without perforation resulting in a generalized or localized peritonitis. Where the inflammation is less acute, adhesions and intestinal obstruction may ensue, Ovens (1943), Waterfall (1948). Perforation may result from infection (Moloney and McQuaid (1949)) but traumatic rupture has also been recorded, Butler (1937).

INTESTINAL OBSTRUCTION. This may follow adhesions due to past or present jejunal diverticulitis, d'Abreu (1944), the impaction of foreign bodies, parasites or to concretions which may enroach on the lumen of the bowel. Acute volvulus is rare as also is chronic volvulus of which a case is described by Porter (1946). Phillips (1953) has drawn attention to the fact that chronic intestinal obstruction may occur with jejunal diverticulosis, with jejunal distension and hypertrophy but where no organic obstructing lesion is present. He suggests that the primary lesion of both conditions is a neuro-muscular one, the diverticula being produced by so-called bowel dyskinesia which accords well with Edwards' original view

HÆMORRHAGE. Hæmorrhage may result from ulceration in jejunal diverticula. This, however, is rare and it must not be assumed too lightly that such bleeding is a result of diverticulitis until other causes have been looked for.

STEATORRHŒA. Jejunal diverticulosis has been found as an occasional cause in cases of steatorrhœa where pancreatic disease has been excluded, Badenoch (personal communication, 1953) The cause of this is unknown but may well be related to the "blind loop syndrome" in which fat absorption is upset by changes in bacterial flora and vitamin content of the small bowel

Treatment. Medical treatment is most likely to be advised for jejunal and ileal diverticula which are diagnosed radiologically and which are not giving rise to serious symptoms A low residue diet and paraffin should be given and strong purgatives avoided. Anæmia and vitamin deficiencies which may result from stasis in the diverticula should be looked for and corrected. Intestinal antiseptics have little place in their treatment When discovered incidentally during laparotomy for some other condition, no surgical procedure is called for unless any of the complications noted above are present When solitary or few in number it would be justifiable to invert the diverticulum into the bowel lumen and reinforce the area with a sero-muscular stitch. In the majority of cases where infection or obstruction of the diverticula has produced symptoms, the diverticula will not have been diagnosed pre-operatively. The segment of bowel giving rise to the immediate emergency must generally be resected but it is not usually possible to eradicate all the diverticula owing to the great length of bowel affected. The most seriously affected piece should be removed, realizing that the disease is a progressive one and that more and larger diverticula may develop in years to come.

tract. Under this definition, in stenosis the obstruction will be partial, while in atresia it will be complete.

To complete the formation of the intestinal tract the hind-gut comes down to join *the proctodeum*, but this is not a simple process. The allantoic stalk comes down to meet the hind-gut and form a common entodermal cloaca. A uro-rectal fold arises from the side walls of the cloaca to divide it into two parts. The anterior portion forming the genito-urinary apparatus, and the posterior *portion forming the rectum*. The posterior portion ends blindly about 2 cm. away from the ectodermal covering of the perineum, but an invagination of the perineum (proctodeum) takes place and joins

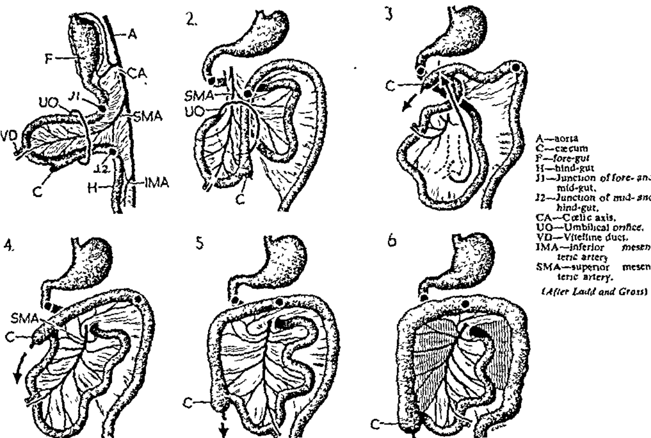


FIG 155. Normal rotation of gut

the blind end separated by a septum, which resolves in due course. Maldevelopment may lead to anal *stenosis* or imperforate anus, and as the genito-urinary apparatus and the rectum are in communication at the cloacal stage it is found that in 50 per cent of cases of imperforate anus there is fistulous communication between the urinary tract and the rectum Maldevelopment of the intestinal tube itself may cause neo-natal intestinal obstruction by stenosis, atresia, and failure of development of the rectum and anal canal, but other causes of obstruction associated *with maldevelopment may be due* to failure of rotation of the mid-gut.

ROTATION OF THE MID-GUT

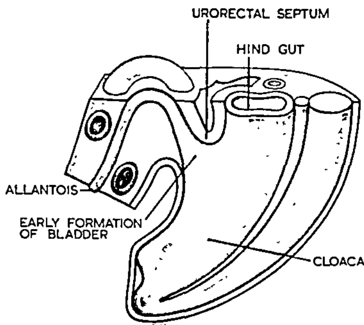
The mid-gut extends from the second part of the duodenum, at the level of the ampulla of Vater, to the mid-point of the transverse colon. At the end of the tenth week the loop

CHAPTER XII

NEO-NATAL INTESTINAL OBSTRUCTION

G. H. MACNAB

THE neo-natal period is defined as the first four weeks of life. In that period the common causes of intestinal obstruction are congenital maldevelopment and malformation. A working knowledge of the development of the intestinal tract from the level of the pylorus



(From "Gray's Anatomy," Longmans Green & Co, London)

FIG 154 To show uro-rectal septum forming to divide the cloaca into two parts The anterior portion assisting in the formation of the genito-urinary apparatus, and the posterior portion assisting in the formation of the rectum

to the anal canal is essential for the proper understanding of the various forms of intestinal obstruction that may arise

Up to the fifth week of intra-uterine life the small intestine from the pylorus to the ileo-cæcal valve is a hollow tube Heaping-up of the lining cells then takes place so that the small intestine becomes a solid cord. This stage passes off by a process of vacuolation so that cystic spaces form and coalesce and reconstitute the lumen by the twelfth week. Information in relation to the large intestine is not so clear, but we do know that areas of atresia can be present, and that until meconium passes through the colon it remains unexpanded with a fine lumen Arrest in the process of vacuolation will set up the condition of intestinal stenosis and atresia In cases of stenosis the lesion is usually limited and consists of a septum with a small perforation in it. In atresia the obstruction may be due to a septum, a length of solid cord, or a length of absent bowel. The atretic area may be single or multiple, or may even extend throughout the length of the intestinal



FIG. 156. Well-formed proctodeum present. Bulging membrane at its base.



FIG. 157. Blind end of hind-gut attached to prostatic urethra.

of mid-gut, which has protruded as a temporary hernia into the root of the umbilical cord, is withdrawn back into the peritoneal cavity. The superior mesenteric artery, running from the abdominal aorta to the summit of the loop (level of Meckel's Diverticulum), divides the bowel into pre- and post-arterial segments. As the bowel is withdrawn into the peritoneal cavity anti-clockwise rotation will take place as the growth of the liver pushes the pre-arterial segment down and to the right, so that the post-arterial segment is forced to rotate to the left and pass upwards. This means that the cæcum, which is represented as a rapidly developing bud on the post-arterial segment, has passed up to the region of the left hypochondrium. Further rotation will cause the cæcum to pass to the right hypochondrium in front of the superior mesenteric artery, from where it will descend into the right iliac fossa. The duodeno-jejunal flexure of the pre-arterial segment, in order to reach the left side of the abdomen, passes behind the superior mesenteric artery. The beginning and the end of the mid-gut are now in close proximity, separated by the superior mesenteric artery, and the dorsal mesentery has only a narrow attachment to the posterior abdominal wall. The final stage of rotation allows the mesentery of the small intestine to widen its attachment, and the mesentery of cæcum, ascending and descending colon to fuse with the posterior parietal peritoneum.

Two common defects giving rise to neo-natal intestinal obstruction are failure of the cæcum to reach the right side of the abdomen, and volvulus of the mid-gut. The cæcum lies in the mid-line, anchored to the right paracolic gutter by a band of connective tissue, which crosses the duodenum and gives rise to partial or complete obstruction In volvulus of the mid-gut the root of the mesentery is narrow and poorly fixed, so that rotation takes place around it, obstructing the bowel, and in some cases, cutting off its blood supply.

The common causes of neo-natal intestinal obstruction due to maldevelopment occur in the following order:

Imperforate Anus
Areas of Atresia and Stenosis in the Small Intestine.
Atresia and Stenosis of the Duodenum.
Volvulus of the Mid-gut.
Failure of Rotation of the Cæcum.

IMPERFORATE ANUS

This condition occurs in about 1 in 5,000 births and 50 per cent of the cases have communication with the genito-urinary tract or the perineum. Intestinal obstruction will develop in the male infant, and if not relieved the infant will die in six days. The female infant usually has a communication with the vagina, so obstruction does not develop till weaning takes place Early diagnosis will influence the treatment, as the cause of death in the neo-natal period is due to loss of fluid and electrolytes into the distended intestine, leading to hæmo-concentration and circulatory failure. In attempting to assess the proximity of the rectum to the perineum it is well to remember that dimpling of the skin in the mid-line, due to the presence of the external sphincter muscle, is not evidence that the rectum is near the surface, as this muscle is formed independent of the intestinal tube Turning of the child upside down and looking for radiological evidence of gas in the blind end can also be misleading, as meconium may have passed down into the blind end, and its sticky character prevent gas rising above it.

proximal to the ampulla of Vater. Stools were passed by 40 per cent of the infants with complete occlusions, and by 80 per cent with incomplete occlusions. Meconium is usually passed within a few hours of birth and milk curds are present in it by the third day. Delay in passage of meconium or change in its consistency associated with persis-



FIG. 158. Plain X-ray of abdomen to show distended loops of small bowel with fluid levels present in a case of atresia of the ileum

tent bile-stained vomiting makes one suspect that intestinal obstruction is present. At this stage Farber's (1933) test will be of value in helping to differentiate between complete and incomplete obstruction. It is dependent on the fact that squamous epithelial cells desquamated from the skin of the fœtus are swallowed in the amniotic fluid in utero and passed in the meconium stool after birth. Absence of these cells denotes complete obstruction. Distension of the abdomen in Louw's series of cases was absent in 20 per cent of cases with incomplete occlusions, and in 50 per cent of cases with complete duodenal occlusions. At the end of 48 hours all infants with complete occlusions of jejunum or ileum will show abdominal distension, and if the distension is

There are only two indications for relieving the obstruction by the perineal route. The first is when there is evidence of definite development of the proctodeum and a bulging membrane is seen in its depths. The second is the presence of a triangular area of skin which forms a flap over a patent anal canal. Cruciate incision of the membrane will restore continuity of the bowel in the first case, and excision of the triangular flap of skin will relieve obstruction in the second. In both cases daily dilatation of the rectum for a period of three months is required, as there is a tendency to stenosis. In all other forms of imperforate anus, giving rise to obstruction in the neo-natal period, laparotomy should be carried out, so that any communication between the hind-gut and the urinary tract can be shut off. In the male, the common finding is the blind end of bowel lying on the abdominal surface of the levator ani muscle attached to the prostatic urethra. This attachment may be in the nature of a fibrous cord, or else a true fistulous communication. Having divided the communication the urinary tract is no longer liable to infection A life-saving colostomy is now carried out, and restoration of rectum and anal canal is delayed until the child is one year old. The colostomy is made through a high right paramedian muscle-splitting incision. The right half of the transverse colon is selected and turned through an angle of 90 degrees. The limbs of the loop are sutured together and skin is brought through the periphery of the transverse meso-colon. The peritoneum of the bowel is sutured carefully to the parietal peritoneum. It is important to take these precautions in the infant, for the colon that is not truly formed usually retains its mesentery. Prolapse through the colostomy is the main complication.

In the early case, 12–24 hours old, I have been carrying out laparotomy and formation of the rectum and anal canal in one stage. The advantages are that gaseous distension has not yet occurred, meconium is sterile so peritonitis does not arise, and communication between urinary tract and rectum can be shut off. Experience shows that at this age the same difficulties arise as occur in operation by the perineal route, namely, that there is tension on the line of anastomosis which leads to stricture formation. When the child is one year old, formation of a functioning rectum and anal canal is dependent on free mobilization of the hind-gut and placing of the anal canal anteriorly, so that it is embraced by the pubo-rectalis sling of muscle fibres forming the anterior part of the levator ani muscle In the male child, this is carried out by forming a channel through to the perineum, which hugs the back of the prostate gland. Excess of bowel must be brought to the perineum and anastomosis made between mucosa and skin of perineum. The colostomy is closed two weeks later by crushing the spur, freeing the bowel from the anterior abdominal wall, and intra-peritoneal closure. Extra-peritoneal closure in the very young may lead to kinking of the bowel. Daily dilatation of the new anal canal must be carried out for the next three months.

Areas of Atresia and Stenosis in the Small Intestine

These areas are commonly found in the jejunum and ileum and tend to be multiple. Stenosis is a rare event and it is always single and gives rise to partial obstruction.

Louw (1952), in an analysis of 76 cases of atresia and stenosis involving the duodenum, jejunum, and ileum, showed that repeated vomiting was the outstanding symptom. It usually commenced on the first day of life, but in 20 per cent of the cases of atresia it was delayed for two or three days, while in 50 per cent of the stenoses it did not start for a week or two. The vomit was bile-stained in all cases except those with occlusion

in the passage of meconium and its consistency should be regarded as suffering from intestinal obstruction until proved otherwise. By the time an early diagnosis has been made the infant is in a debilitated condition due to the fact that the intestinal secretions have been retained in a distended bowel in utero, so that early vomiting causes rapid loss of fluid and electrolytes, which in turn causes hæmo-concentration and alteration in the bicarbonate reserve of the blood. The infant soon shows evidence of dehydration



Fig 160. Distended proximal end of small intestine in a case of atresia.

and peripheral circulatory failure. Instability of the respiratory centre leads to atelectasis, and vomit passes easily into the lungs, leading to broncho-pneumonia

Treatment of atresia and stenosis of small intestine is by early laparotomy. Electrolyte and fluid loss is restored by intravenous infusion, and the fluid content of the obstructed intestine is aspirated through a rubber catheter placed in the stomach Intratracheal anæsthesia is used so that a high oxygen content can be delivered, and the complication of spasm of the glottis avoided In addition, the intra-tracheal tube prevents inhalation of fluid content which may be forced up around the gastric tube during manipulation of the intestine by the surgeon. A long right paramedian incision is made as adequate exposure is essential. The intestines are delivered out into the wound

severe and associated with the presence of dilated veins on the abdominal wall and generalized cyanosis, perforation, and peritonitis should be suspected. Plain X-rays of the abdomen in the prone and erect positions will give evidence of distended bowel and fluid levels in complete occlusions, but they may be absent in incomplete occlusions. It is well to remember that air will pass from the stomach to the colon in 6 hours in the normal new-born infant, and coils of small intestine are often distended with air



Fig 159 Multiple areas of atresia of small intestine

giving the appearance of obstruction The presence of fluid levels confirming complete obstruction are difficult to detect if areas of atresia are multiple, as distended loops of small intestine are superimposed on each other. It is also well to remember that until meconium has entered the colon, it will remain in a state of microcolon, so that the absence of air is not evidence of atresia of the colon. X-rays may assist in localization of the obstruction, but clinical signs will decide for or against laparotomy. Early diagnosis is essential to successful treatment so that any new-born infant suffering from repeated bile-stained vomiting associated with abdominal distension and abnormality

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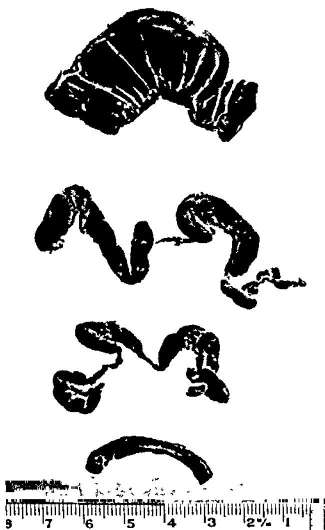


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Fig. 161. Duodenal atresia—Note the large dilated segment of duodenum proximal to the area of atresia.



Fig. 162. Duodenal atresia—showing barium in stomach and in the distended duodenum.

and covered with warm, wet pads. The assistant must support the intestines to prevent any drag on the mesentery. Careful inspection of the whole length of the bowel is essential as there may be multiple areas of atresia, even involving the colon. A single area of atresia can be resected, but if there are multiple small areas throughout the small intestine a short-circuit operation is carried out, and the closed loops of poorly-formed bowel are removed at a later date. A single area of atresia is resected and anastomosis made along an oblique line with a single layer of interrupted silk sutures. It is essential to excise the blind end of the proximal segment before carrying out anastomosis, as distension over a long period has set up venous congestion and anoxia in the bowel wall, leading to potential gangrene. Disparity between the proximal and distal lumens of intestine can be overcome, to a slight extent, by distension of the distal end with saline. Recent experimental work in young animals has shown that the technique of end-to-end anastomosis has no advantage over side-to-side anastomosis, except that it is a more simple procedure. The chief complication is post-operative paralytic ileus, which is due to the proximal bowel having been grossly distended for a long period of time, and the distal bowel having not yet been dilated by meconium, which has been held up and become viscid in character. Attempts have been made to overcome this complication by carrying out double enterostomy of proximal and distal bowel. Daily dilatations of the distal bowel with saline solution is carried out, and continuity of the gut restored at the end of five days. This procedure is unsatisfactory owing to excessive electrolyte and fluid loss. In cases of direct anastomosis dilatation of the colon with repeated enemas is of little value, as it exhausts the infant and may lead to perforation of the bowel. In cases of paralytic ileus of this type the bowel may not act for six days, so that the surgeon, during this time, must not yield to the temptation to subject the infant to a second operation, to see that all is well with the anastomosis. Stenosis occurring in the small intestine involves a single area and is best dealt with by resection as the opening is usually small, and in the case of obstruction due to a diaphragm, perforation of it gives no guarantee of an adequate opening.

Atresia and Stenosis of the Duodenum

Louw (1952) has shown that 40 per cent of the cases of atresia and stenosis occur in the duodenum. One-third were situated at, or just proximal to, the ampulla of Vater; one-third just distal to the ampulla, and one-third in the region of the duodeno-jejunal flexure. Duodenal stenosis is a rare event and consists of a diaphragm of tissue with an opening in it placed at the level of the ampulla In atresia, when the block is complete, there is gross dilatation of the stomach and proximal duodenum with incompetence of the pyloric sphincter. The average duration of life in severe cases of atresia and stenosis is five to six days, but the rare cases of stenosis may survive up to seven years.

The clinical picture is one of repeated vomiting from the first or second day of life. If the obstruction is distal to the ampulla the vomit will be bile-stained, and in 40 per cent of cases projectile in character. Visible gastric peristalsis is only seen in a small percentage of cases, as the pyloric sphincter is incompetent, leading to gross distension of stomach and duodenum. Plain X-ray examination will show air outlining the distended stomach and duodenum, and in atresia it will not be present in the small intestine. Air can be aspirated from the stomach and duodenum by passage of a gastric tube and

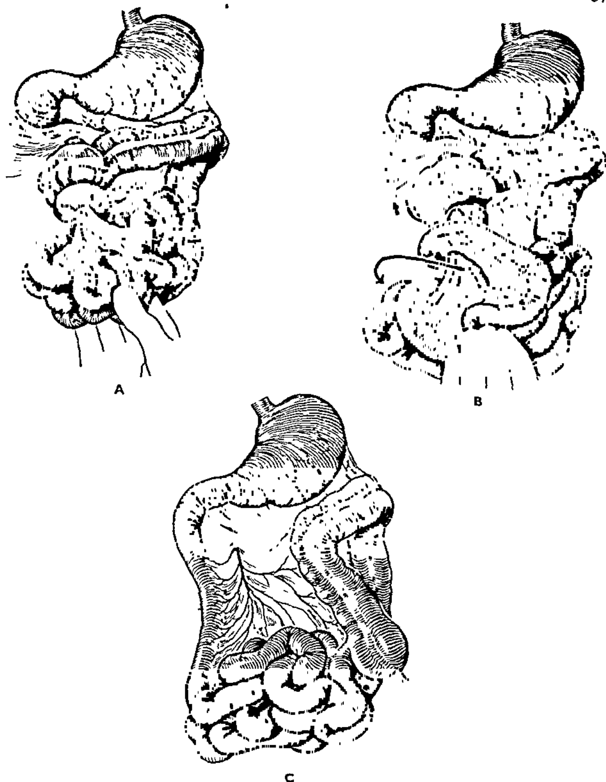


FIG 163 Duodenal compress [illegible]

(a) This [illegible]

(b [illegible]
volvu [illegible]

(c) The [illegible] has been divided and the cæcum placed to the left of the mid-line and the volvulus of [illegible] to undo the the small intestine reduced

(*After Ladd and Gross "Abdominal Surgery of Infancy and Childhood"* 1941)

a second X-ray will confirm the diagnosis. If doubt remains a thin barium solution can be run into the stomach and X-ray will show the distended duodenum appearing like a second stomach. Following X-ray examination the barium solution must be aspirated as these weakly infants are inclined to regurgitate and inhale the vomit. The passage of stools will vary from nil to a small hard motion mixed with mucous.

Treatment. Duodeno-jejunostomy is the operation of choice for dealing with duodenal stenosis or atresia, but if the block can be proved to lie proximal to the ampulla then anterior or posterior gastro-jejunostomy can be carried out. In the operation of duodeno-jejunostomy the size of the duodenum is satisfactory, but the thinness of its walls makes suturing a difficult procedure. A side-to-side anastomosis with a single row of interrupted silk sutures is made between the dilated duodenum and the first coil of jejunum

The post-operative complications of paralysis of the proximal distended segment with gastric retention is a common cause of death. Continued tension on the suture line may lead to a leak and peritonitis, and failure of peristaltic movement causes vomiting to persist, resulting in inadequate nutrition so that the weakly infant may inhale his vomit and die from aspiration broncho-pneumonia. Ehrenpreis and Sandblom (1949) have devised a method to overcome these complications, and have reduced their mortality since 1945 from 75 per cent to 25 per cent. Before completing the duodeno-jejunostomy they make an opening into the stomach and pass a tube through the stomach, pyloric canal, and anastomosis, into the efferent loop of jejunum. The tube and stomach are fixed to the anterior abdominal wall. Contents can be aspirated and the infant fed past the anastomosis until sound healing and restoration of peristaltic movement has taken place When the tube is removed the gastrostomy opening will soon close over. It is very difficult in these infants to guide a tube from the mouth through the anastomosis, and in addition, an indwelling catheter is liable to set up pharyngeal irritation.

VOLVULUS OF THE MID-GUT

This condition is encountered in the new-born and is due to failure of rotation in its final stage so that the mesentery of the small intestine does not gain a wide attachment to the posterior abdominal wall. It will be remembered that the beginning and the end of the mid-gut are placed close to each other and separated by the superior mesenteric artery, so a pedicle is formed around which rotation can take place. The small intestine rotates in a clockwise manner so that obstruction occurs at the level of the duodeno-jejunal junction, and as rotation proceeds the superior mesenteric artery and its branches are compressed so that the blood-supply to the bowel may be cut off and gangrene and peritonitis develop. The clinical picture is one of a dehydrated infant with repeated bile-stained vomiting and a distended abdomen If strangulation has occurred melæna may be present

Treatment consists of early laparotomy. The volvulus is untwisted by rotation in an anti-clockwise manner, and the bowel inspected for evidence of gangrene. If gangrene is present the affected segment has to be resected. The mesentery of the small intestine should then be fixed to the posterior abdominal wall with interrupted silk sutures in order to give it a wide attachment.

urine cannot be accurately measured, but diminution in volume can be observed by the nurse who is changing the napkins. Urine can be tested for the presence of or absence of chlorides by Fantus's Test (1936) as only 10 drops of urine are required. The normal concentration should exceed 1 gm. per litre. Isolated estimations of Hæmoglobin, Red Cell concentration, or serum protein levels are of little value, as there is a wide range of the normal in the first few months of life. Persistent high blood urea and non-protein nitrogen levels are a constant feature of dehydration but will return to normal within 48 hours, provided that intravenous therapy is satisfactory and a free flow of urine is established. If the blood urea remains high under these conditions kidney damage should be suspected. In severely dehydrated infants estimation of the carbon dioxide combining power and chloride levels of plasma are of value in guiding treatment. In alkalosis the carbon dioxide combining power is high and the chloride level is low. In the treatment of neo-natal intestinal obstruction the intravenous route should always be used as rapid restoration of volume of body fluids is required as well as restoration of the electrolyte balance. The daily volume of fluid required by an infant is 2½ oz. per lb. of body weight, and when dehydration is present 2·5–5 per cent of the body weight should be added to the fluid volume. As a method of resuscitation a solution of 5 per cent glucose in 0·45 per cent sodium chloride should be given rapidly up to a maximum of 10 ml. per lb. of body weight. When the vomiting is severe 5 per cent glucose in isotonic saline solution should be substituted. The initial infusion can be followed by a mixture of plasma and 10 per cent glucose solution in equal parts, which should be given at the rate of 5–10 ml. per lb. per hour for a period of up to 4 hours. As soon as urine has been passed the rate of infusion can be reduced to 3–4 ml. per lb. per hour. This means that the infant will receive a total of 90–130 ml. per lb. in the first 24 hours. When sounds of peristalsis return gastric suction may be terminated and oral feeding resumed. If the infant will not tolerate oral feeding, then daily allowance of protein in the intravenous infusion must be provided at the rate of 1 gm. of protein per lb. per 24 hours in the form of plasma or hydrolysed casein. In these desperately ill small infants the position must be reviewed at 6 hourly intervals and the nature and rate of the infusion changed according to assessment. The infant must not be heated up until the fluid volume is restored, as peripheral vasodilatation would further embarrass the poor circulation.

References

Louw, J. H (Sept. 1952) *S. A J. Clin. Science.* 3, 109–129.
Farber, S J. (1933) *Amer. Med. Assoc.* 100, 1753.
Ehrenpreis, T. L. and Sandblom (1949) *P. Acta Paed. Scand.* 38, 109.
Ehrenpreis, T L. (1946) *Acta. Chir Scand.* 94, Suppl. 112,
Nixon, H H (Sept. 1953) *Brit. J. Surg.* 41, 184–188.
Farber, S J. (1944) *Pediat.* 24, 387
Farber, S J. [illegible]
[illegible]
[illegible]
[illegible]
Thorndike, A., Jnr. and Ferguson, C. F. (1938) *Am J. Surg.* 39, 429.
Fantus, J. B. (1936) *J. Amer. Med. Ass* 107, 14.

Treatment. This consists of early laparotomy, as the viscid meconium distends the ileum, and its weight may cause the terminal loop to undergo rotation. The findings at operation are as follows. The level of the obstruction is usually in the lower ileum. As you trace the bowel down to the level of obstruction it not only dilates but becomes hypertrophied, suggesting that chronic obstruction has been present in fœtal life. The lowest loop is distended and heavy with meconium, so volvulus may be present leading to gangrene and peritonitis. The bowel tapers from the distended loop by a conical segment to the unexpanded bowel. The meconium in the obstructing loop consists of firm putty-like material, which sticks to the fingers on attempting to milk it through an opening made in the bowel. A satisfactory method of operative procedure is as follows:

The apex of the lowest distended loop should be opened and a small Paul's tube tied in. The bowel is then fixed to the anterior abdominal wall. Fifteen per cent pancreatin solution is instilled twice a day until the meconium plug is dissolved. In Nixon's (1952) case meconium was passed per rectum on the fourth day and oral feeding was started Fifteen grs. of pancreatin granules (triple strength) were added to each feed and Amigen given to supply extra protein. Owing to the viscosity of the meconium, fluid and electrolyte loss from the enterostomy is not a problem. As soon as normal stools are being passed the enterostomy can be closed and replaced inside the peritoneal cavity. Pancreatin granules have to be given by mouth throughout the childs' life, otherwise the typical offensive stools of fibro-cystic disease of the pancreas will form

When we turn to the extrinsic causes of neo-natal intestinal obstruction, inguinal hernia accounts for 17 cases out of 118 (Louw, 1952). Thorndike and Ferguson (1938) showed that in 1740 operations for inguinal hernia in childhood, 6 per cent had incarcerated or strangulated herniæ. Incarceration is the common form of obstruction in the neo-natal period, and in 80 per cent of cases the hernia can be reduced by conservative measures. An ice-bag is applied to the swollen area to reduce œdema and pethidine is given to induce sleep and relax the abdominal wall. This treatment can be persisted in up to a period of 3 hours

Internal herniation accounts for a very small percentage of cases of obstruction, and is usually due to congenital defects in the mesentery. On opening the abdomen it is important to make sure that the condition is not due to malrotation; for coils of intestine twisted on a pedicle give the appearance of intestine passing through an opening in the mesentery.

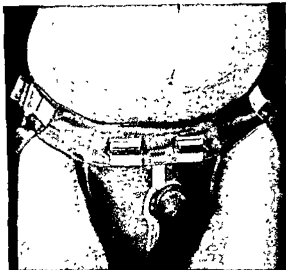
Diaphragmatic hernia due to persistence of the pleuro-peritoneal canals will allow the small intestine to pass into the chest, where kinking of the bowel can give rise to acute obstruction, but the signs are overshadowed by the degree of respiratory and cardiac embarrassment

In treatment of neo-natal intestinal obstruction the maintenance of fluid and electrolyte balance is of the greatest importance. This subject is fully dealt with in Chapter IX, but there are one or two points of difference in the neo-natal infant. Infants become more rapidly dehydrated than adults, due to the fact that the metabolic rate of the infant is greater than that of the adult, and losses in water are proportional to the metabolic rate. The kidney of the infant in the first 2 months of life is unable to fully concentrate its constituents for elimination, so large quantities of urine have to be passed. In severe dehydration an infant may lose up to 15 per cent of body weight. Loss of weight associated with oliguria is the first clinical sign of dehydration. Fall in the output of

Diagnosis. An external hernia causes a swelling in an anatomical position known to be either a congenital point of potential weakness or a probable weak point following trauma, surgical intervention, or acquired disease. Certain normally "external" herniæ may not however be clinically evident in this way—a Richter's type of femoral hernia being one example.

The swelling is usually palpable, and commonly has an expansile impulse on coughing; but if complicated by inflammation, irreducibility or strangulation will not have such an impulse. The swelling will often be reduced spontaneously when the patient lies down, or the herniated viscera may be returned to the peritoneal cavity leaving





(*From "Hernia" by F Mitchell-Heggs, J & A Churchill Ltd, London*)

Fig 165 Irreducible scrotal hernia, fitted with bag truss Provision for shoulder braces if required

the sac *in situ* by manipulation or taxis. The path of descent of each hernia tends to be controlled by the anatomy of the particular area and it therefore takes place along a probable known route.

An internal hernia only occasionally causes local signs—an obturator hernia and diaphragmatic hernia being generally exceptions to this rule. They commonly become evident as a form of acute intestinal obstruction, the diagnosis being established following laparotomy.

Reduction. This is usually brought about by manipulation, but, failing this, operative reduction following surgical exploration must be undertaken. In very large diaphragmatic and epigastric hernias manipulation is often impracticable or inadvisable.

Treatment by Apparatus. Following reduction, it will be necessary to prevent a recurrence of the protrusion. In cases where operation for medical or personal reasons is considered inadvisable resort must be made to palliative treatment by a truss or abdominal support.

Such treatment should be carried out whenever possible with the assistance of a certificated member of the Truss Fitters Group of the Institute of British Surgical Technicians. These trained assistants to the profession have passed an examination in anatomy and practical truss application following special courses of instruction under a syllabus devised by the Royal College of Surgeons. Further details of this type of

CHAPTER XIII

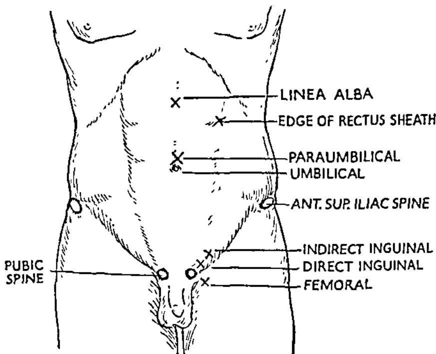
HERNIA

F. Mitchell-Heggs

ABDOMINAL HERNIA

Definition. An abdominal hernia is a protrusion of abdominal contents. Inguinal, femoral, and umbilical herniæ are almost invariably enclosed within a peritoneal sac, but certain diaphragmatic, sliding inguinal, supravesical, and incisional herniæ are not.

Classification. Abdominal contents may protrude anteriorly causing epigastric, Spigelian, umbilical or incisional hernia or they may appear in the inguinal or femoral



(From "Hernia" by F Mitchell-Heggs, J & A Churchill Ltd, London)

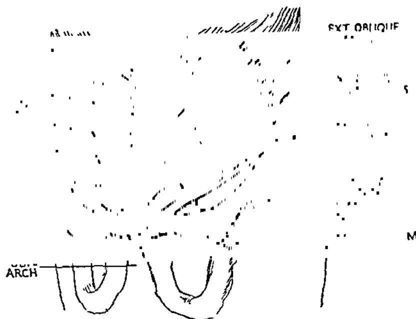
Fig 164 The anterior abdominal wall and its main sites of hernia

region. Superiorly a diaphragmatic hernia will result and inferiorly, perineal, sciatic, and obturator hernia. The posterior abdominal wall is the site of lumbar hernia and is related to the concealed herniæ which arise in the duodenal, mesenteric, cæcal, and other intraperitoneal sacs

Causation. The majority of herniæ are congenital in origin. Indirect inguinal, developmental diaphragmatic and umbilical hernia in children are typical examples. Incisional hernia whether due to intra-abdominal sepsis or following damage to the nerves or muscle layers of the abdominal wall and diaphragmatic hernia after disease or injury are typical acquired varieties The causation of para-umbilical hernia in adults, epigastric hernia and Spigelian hernia is probably due to fatty deposition whereas interaponeurotic and femoral herniæ are probably congenital in origin.

prolonged (21 days) bed rest with full mobility in bed under the care of the physiotherapist. The type of suture material used, the operation undertaken and the patient's general constitution are controlling factors. My own preference is towards early ambulation combined with the use of non-absorbable suture material.

Light work may be commenced about 4 weeks after the patient has got up, but heavy work should either be forbidden or delayed for between 3 and 6 months in the more severe cases. All hernia cases should be followed up at intervals for several



(From "Hernia" by F. Mitchell-Heggs, J & A. Churchill Ltd, London)

FIG. 167 To show the attachment of the external oblique muscle and aponeurosis to the pelvis.

years in order to exclude recurrence. Recent work has shown that recurrence rates of hernia operations calculated on a basis of less than 5–10 years follow up are inaccurate.

Obese patients will continue to require dietetic control, and those with weak muscles will need either physiotherapy with or without a surgical support or a light flat-padded post-operative truss, depending upon the patient's constitution and background.

INDIRECT INGUINAL HERNIA

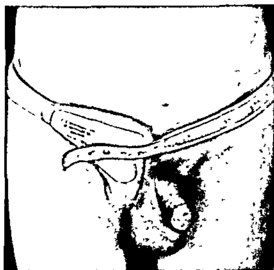
Causation. This is congenital. These herniæ are the result of failure of obliteration of the proximal part of the processus vaginalis testis. The immediate onset may be brought about by any condition giving rise to a sudden increase in intraperitoneal pressure occurring at a time of unguarded muscular activity. The protrusion follows the path of the sac within the spermatic cord from the internal abdominal ring, through the inguinal canal to the external abdominal ring and down towards the testis. In the female it passes along the inguinal canal close to the round ligament.

Classification. Such a hernia confined within the inguinal canal is a bubonocele. Interval grades from this type to a complete hernia, which passes to the testicle in the scrotum, are found. The hernia may contain omentum (epiplocele), small or large intestine, the appendix, or other viscera and an associated second hernial sac may be found in any of the abnormal directions known to occur with a misplaced testicle:

treatment are laid out in the manual on Truss Fitting which has been specially compiled for their training.

Pre-operative Treatment. Before an operation is considered, the general health of each patient must be studied, the presence of multiple herniæ noted and any associated disease investigated. Probable causes of post-operative raised intraperitoneal pressure—respiratory, alimentary, urethral, must be investigated and treated, and the age, build, occupation, and other factors considered of each patient. Treatment for these associated





(From "Hernia" by F Mitchell-Heggs, J & A Churchill Ltd, London)

Fig 166 Spring-type, Semi-scrotal Notched pad Truss for right inguinal hernia

complications may be necessary and are almost as important as the surgical manœuvre itself. The postponement of an operation until the summer, breathing exercises and postural drainage, treatment of a stricture or prostatectomy and weight reduction are cited as common examples.

Anæsthesia. Here it is more important to have an expert anæsthetist, particularly in senile and difficult cases than to have a preferred method. Good oxygenization of the blood, a safe depth of anæsthesia, the patient's comfort and wishes and adequate relaxation are the essentials In some clinics a particular method or preference for one is common Local anæsthesia, epidural block, spinal anæsthesia and most of the varieties of general anæsthesia are all advocated In hernia associated with intestinal obstruction and dehydration, the use of gastric suction and intravenous infusion both before, during, and after the operation are of proved value

Operative Treatment. Removal of the hernial sac is the basic aim, followed when necessary by alteration of the existing anatomy of the part by suture or the insertion of some autogenous graft or exogenous foreign body into the weakened area of protrusion. When complications such as intestinal obstruction, strangulation, or gangrene are present these are treated and the hernia, together with any associated disease such as incomplete testicular descent, dealt with later

Post-operative Treatment. Breathing exercises, and early mobility in bed are the important considerations. In recent years the tendency has been towards early (2 day) ambulation but surgical opinion is not yet agreed upon this Many surgeons advise

ring. The ring itself need in many cases not be opened. The spermatic cord is exposed as it lies in the inguinal canal and the ilio-inguinal nerve is seen passing obliquely from above downwards between the external and internal oblique muscles on its way to join the spermatic cord. Its position is noted and care is taken not to injure it.

The cremasteric fascia is picked up by tissue forceps and incised for one inch. This opening is further enlarged by blunt dissection and the incision may be increased in length as required until the position of the sac and its white texture is recognized. The sac lies anteriorly in the spermatic cord. It is picked up with hæmostats and gradually separated from the other constituents of the cord by gauze or blunt dissection, aided when necessary by sharp dissection with the scalpel.

The sac is dissected out and exposed as far as its neck at the internal abdominal ring—which position is often shown by a pad of fat adherent to the peritoneum at that point. The hernial contents will usually have been reduced at this stage of the operation but when they are adherent, the sac is opened, usually at the fundus and at a safe part and the adhesions preventing reduction are cut. The sac is opened at the fundus and a finger is inserted down to the internal abdominal ring.

The Sac Stump. It is usual to twist the neck of the sac in order that, before transfixing it with a ligature, no viscus should lie within it. The sac after transfixion and ligature is cut off and removed. If it is considered that the neck of the sac is a potential weak point for recurrence, the stump may then be fixed by "the stump fixation manœuvre." The ends of the stump ligature are left long and are separately re-threaded on to a hernia needle. The needle is then passed through the conjoined tendon or transversus muscle from the deep surface superficially. The two separate ends which now appear on the surface of the muscle are tied in such a way as to pull the neck of the sac upwards and outwards and to fix it to the under surface of the transversus muscle. Care should be taken not to include the ilio-inguinal nerve in the ligature.

Inguinal Ring Herniotomy. In infants, children, and young adults, no further hernial repair is necessary than removal of the sac. When it is considered, in adults, that the internal inguinal ring, middle inguinal ring (of Lytle) or transversalis fascia have been unduly stretched, it is usually advisable nowadays further to strengthen the posterior inguinal wall and ring. To demonstrate the ring, the sac is opened and a finger inserted into its neck. Torsion on the sac will reveal adherent fascia which can be gently dissected off to show the margins of the ring beneath. The position of this is further gauged by the flexed index finger feeling from within the sac and pressing against its rim. Having displaced the spermatic cord upwards and ligated the sac remnant, the margins of the internal ring are defined and held up by suitable tissue forceps. Several simple or mattress sutures are then placed between the pillars of the ring. This operation of ring herniotomy has been stressed in recent years by Lytle (1945).

Transversalis Herniotomy. Tightening of the transversalis fascia has been an integral part of the Bassini type of operation for many years. Although ring herniotomy is advocated by many surgeons, Monro (1953) has pointed out that the *repair of the* internal ring alone by taking a tuck in its inner margin is unsound. He compares the effect of abdominal pressure on the ring and transversalis fascia with that of the wind filling a sail. He therefore considers that to repair such a hole in a sail by a tuck would be a bad principle and strongly condemns plastic repair of the ring both from the theoretical and from the post-operative angle. He advises that the whole transversalis fascia

e.g. the perineum, thigh, suprapubic region or right iliac fossa. A direct or a femoral hernia may also be present or the sac may drag either the bladder, the cæcum or the pelvic colon into its wall (hernia-en-glissade).

Diagnosis. The swelling appears at the external abdominal ring and may pass down into the scrotum (scrotal hernia)

Differential Diagnosis. Hydrocele of the tunica vaginalis testis, hydrocele of the cord, spermatocele, varicocele, inguinal abscess, inguinal glandular enlargement and abnormally descended testicle should be excluded.

Complications. The hernia may become inflamed, obstructed, irreducible, or strangulated. Independent pathological conditions (tuberculous peritonitis, acute appendicitis) may affect the peritoneum, the peritoneal fluid, or the visceral contents of the hernia.

Reduction. The patient should lie down on a couch with the head and shoulders and the spine and hip joints flexed. The mind and body should be relaxed. The neck of the sac is gently held by the fingers of one hand while the contents are slowly and carefully returned to the abdomen by a light squeezing movement together with pulsion towards the external abdominal ring. Force is never used. A characteristic gurgling sound and the gradual disappearance of the swelling will denote a successful reduction.

Maintenance of Reduction. This is by pad and bandage, truss, abdominal support, injection therapy, or operation. The causes of repeated coughing and straining must be treated and removed.

Truss Treatment. This is undertaken when operation is either not advised or is refused. In adults a tempered spring, leather covered truss with an adequate firm pad is normally prescribed. An elastic truss is an alternative, particularly in thin and older patients, but it is considered to be less reliable.

In children a horseshoe-shaped solid rubber double truss which is washable is found more practicable. Pneumatic and soft pads and any variety of truss with a mobile head are not advised. Celluloid covered trusses are preferred by some patients owing to their improved cleanliness.

Injection Treatment. A sclerosing substance can be injected into and around the *sac of the hernia. The manœuvre is relatively blind and anatomically dangerous.* Follow up observations of this type of treatment have not been satisfactory and it may be regarded nowadays as obsolete

Inguinal Herniotomy. In this operation the hernial sac is removed. Of historical interest only are the low and intermediate excision herniotomy procedures in which the sac was removed at the level of the external abdominal ring and within the inguinal canal respectively.

High Ligation Excision Herniotomy. This is the basis of all modern hernia operations. The sac is regarded as a congenital abnormality and its removal, particularly in infants, children, and young adults, does in fact almost invariably lead to a permanent cure.

The inguinal canal is exposed by an incision extending from just above the pubic spine to the upper margin of the internal abdominal ring. It is made ¾ in. above and parallel to the line of the inguinal ligament The skin and superficial fascia are incised in the same line and the branches of the superficial epigastric vessels are exposed thereby, ligated in continuity and cut across as they pass through the line of incision. The deep fascia is incised and the external abdominal ring exposed. The external oblique aponeurosis is then cut in the line of its fibres commencing just above the external abdominal

ring. The ring itself need in many cases not be opened. The spermatic cord is exposed as it lies in the inguinal canal and the ilio-inguinal nerve is seen passing obliquely from above downwards between the external and internal oblique muscles on its way to join the spermatic cord. Its position is noted and care is taken not to injure it.

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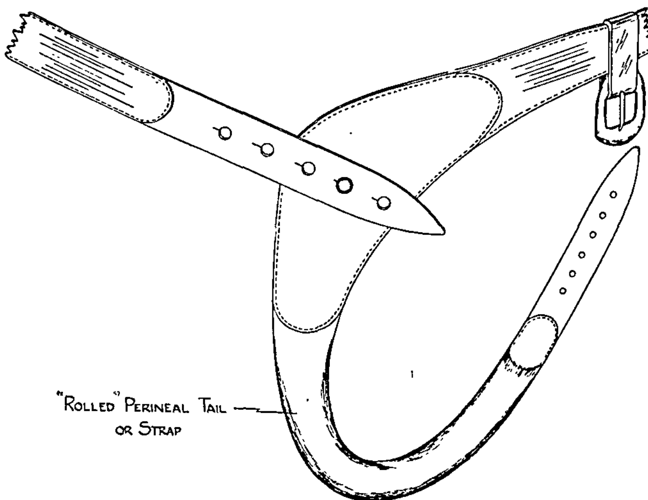
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should be repaired as a sheet in order to equalize tension within it and considers that it should be sutured, as in the original operations, to the inguinal ligament.

In other clinics, doubt has been expressed as to the value of placing any reliance on suture of a fascia which it will be admitted is in many cases friable and thin and in other cases infiltrated with fat.

Lytle has drawn attention to a second lower and wider ring which often occurs in moderately large hernial protrusions. He has named this the *middle inguinal ring* and has advised that when it is present it should be sutured separately.



(From Hernia by F Mitchell-Heggs, J & A Churchill Ltd, London)
Fig 168 A Rat-tail inguinal truss

High Ligation Orchidectomy. In an old man, particularly when the hernia is very large and either obstructed, strangulated, or irreducible, it may be wise, following reduction, to perform an orchidectomy This operation may subsequently be followed by one of the herniorrhaphy or hernioplasty procedures. The recurrence rate following this type of operation is very low

Hernia-en-glissade. When, following exposure of the hernial sac as previously described, it is found that bladder, cæcum, or pelvic colon form part of the wall of the hernia, the operation of high ligation excision herniotomy will be impossible. A grid-iron type of incision for colopexy as a supplement to the repair of the inguinal canal has been in use for many years; but it was La Roque (1919) who advocated this type of approach

for inguinal herniotomy as a routine. Nowadays an accessory grid-iron incision is made through the muscles of the abdominal wall, following retraction of the previous inguinal incision, when a hernia-en-glissade is encountered. This allows the surgeon to see the hernia from within the peritoneal cavity and to reduce the sac and the protruding viscera aided by vision from above. It will then be found that the part of the hernial sac which has been previously opened in establishing the diagnosis has in a way, turned itself inside out to finish up as the pelvic mesocolon or other part of the parietal or visceral peritoneum. The defect can then be sutured under direct vision safely. The region of the internal abdominal ring and posterior abdominal wall will then be repaired by a herniorrhaphy or hernioplasty operation.

Double Inguinal Hernia. In some clinics operations upon both inguinal canals at the same time are sometimes undertaken. Although this means only one operation for the patient, post-operative chest complications are more likely to follow because the patient is less able to contract his abdominal muscles when coughing. Also the chance of recurrence is considered to be higher as are the possible subsequent dangers of wound infection resulting in thrombosis.

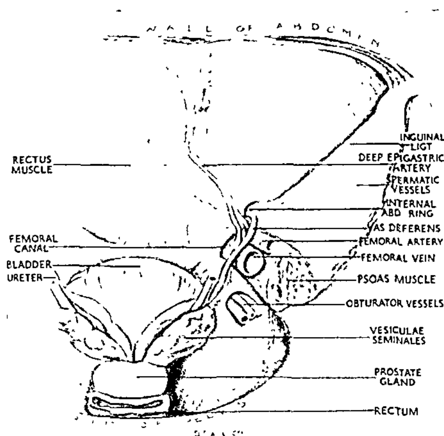
Moderate and Large Inguinal Herniæ. After the hernial sac has been removed, and any necessary repair of the fascia undertaken, it will be found, in moderately large, and large herniæ, that the shutter mechanism of the inguinal canal has been for practical purposes stretched and destroyed. It is for this type of hernia that the herniorrhaphy and hernioplasty procedures are undertaken. Much criticism has been levelled against the Bassini, Halsted, and Ferguson operations because it is thought that they prevent the inguinal shutter mechanism from working normally. These operations are therefore not performed in infants, children, or young adults

Herniorrhaphy. In this type of operation the spermatic cord is transplanted and certain muscular and aponeurotic layers are sutured together to provide a fibrous block across the area of protrusion. The repair is usually related to Poupart's ligament and either the conjoined tendon of the internal oblique and transversus muscles or the rectus sheath are joined to it. The classification of such operations is made according to the position of the spermatic cord, which may be either superficial to, included within or deep to the external oblique aponeurosis.

Inguinal Herniorrhaphy	(1) Subaponeurotic—transversus	Bassini 1889 Marcy 1881. Tanner 1942.
	(2) Extra-aponeurotic	Halsted 1889.
	(3) Interaponeurotic	Andrews 1893.
	(4) Transfemoral (obsolete)	Cheever 1923
	(5) Retroperitoneal (obsolete)	Fowler 1897.
Iliopectineal Herniorrhaphy		Lotheissen 1898. McVay. Anson 1949.
Rectus Herniorrhaphy		Bloodgood 1918.

Since these operations have been carried out by many surgeons all over the world there has been ample time and opportunity to investigate the chance of recurrence in

herniorrhaphy. Charles Mayo (1949) was forced to conclude that there is no perfect surgeon and no perfect surgical procedure in this respect. The recurrence rate has been estimated by Tanner to be 20 per cent, by Edwards (1943 British Army) to be 12 per cent, by Sir Max Page to be 20 per cent, by Clear to be 11 per cent and by Mayo to be 11·9 per cent. Some of these reported series were personal and some were reports and conclusions based on the work of others. They include Bassini, Ferguson, Halsted, and other herniorrhaphy procedures and the period of follow up varies between 3 and



(From "Hernia" by F Mitchell-Heggs, J & A Churchill Ltd, London)

FIG 169 Posterior aspect of anterior abdominal wall and inguinal region

10 years. One conclusion to be reached from the follow up recurrence rates is that no group of figures are of value as a final estimate of recurrence under at least 5 and preferably 10 years' observation.

Subaponeurotic Transversus Herniorrhaphy. Although Bassini's operation was first described in 1887, it is interesting to note that, at a discussion meeting of the surgical section of the Royal Society of Medicine in London in 1952, the operation was advocated by many for the repair of moderate and larger inguinal herniæ. It is in fact generally applicable, involves little retraction, and avoids the danger of injury to the femoral vein. No foreign body apart from the ligatures is included, and the spermatic cord is not left in a subcutaneous position exposed to trauma.

The incision and exposure are as described for high ligation excision herniotomy, but it is necessary in every case to open the external abdominal ring. The exposure and

suture of the internal abdominal ring or transversalis fascia are as described under ring and transversalis herniotomy. The cord, since it is to be transplanted, must be freed by dissection and lifted out of the wound. It is held in this position by a·gauze swab retractor.

The glistening silvery white abdominal surface of Poupart's ligament is exposed and cleaned and the pubic spine is felt for and identified. A series of sutures is then placed at centimetre intervals between the conjoined tendon and Poupart's ligament behind the spermatic cord. The first suture includes the periosteum over the pubic spine and closes the medial end of the posterior inguinal wall—a common site of recurrence. Tension on the sutures should be as little as possible because this may result in the stitches cutting out. The suture material may be catgut, silk, Mersiline, steel wire or other material chosen by the surgeon. The spermatic cord is then replaced and the external oblique, deep fascia and skin sutured in layers.

Extra-aponeurotic Transversus Herniorrhaphy. The Halsted principle also includes high ligation excision herniotomy and repair of the transversalis fascia. The operation proceeds on the lines of the previous herniorrhaphy, but differs from it in that the spermatic cord is placed anterior not only to the sutured surfaces of the conjoined tendon and Poupart's ligament but also to the external oblique aponeurosis. In other words the external abdominal ring becomes newly placed and lies anatomically above the position of the internal abdominal ring.

Inguinal Transversus Herniorrhaphy. Tanner's slide operation has given good results in the treatment also of direct inguinal, recurrent, and strangulated hernia. It has also been used in the surgical treatment of femoral hernia. This operation can be regarded as a modification of the Bassini repair in that the conjoined tendon is joined behind the spermatic cord to the inguinal ligament. There is, however, no tension on the stitches at the site of union because a relief incision is made in the internal oblique and transversus part of the anterior rectus sheath.

An inguinal incision is made, the inguinal canal exposed and hernial sac dissected out and removed When the repair is to be performed, the external oblique part of the anterior rectus sheath is separated and lifted away from the deeper fibres derived from the internal oblique muscle. The incision in the anterior rectus sheath extends from the pubic crest upwards and outwards to a level one hand's breadth above the pubis. It terminates just medial to the edge of the rectus sheath, so that when the slide has taken place, the rectus muscle fills the gap so made. It is important to avoid injury to the ilio-hypogastric nerve which lies close to the line of incision.

As in other forms of herniorrhaphy this operation is not undertaken unless it is considered that the normal internal abdominal ring and shutter mechanism have been destroyed by stretching. The method is increasing in popularity. In Tanner's series 116 cases were dealt with, with only one bulge type of "recurrence"; of these cases 38 were over the age of 60, 42 were for strangulated hernia, 20 of them direct hernia, and 4 for recurrent hernia.

Ilio-pectineal Transversus Herniorrhaphy. McVay and Anson, Donald, Rains, and other workers consider that it is incorrect anatomically to join the conjoined tendon to Poupart's ligament in the repair of inguinal hernia. Their conclusion following many dissections, is that a failure of development in both strength and attachment of the transversus aponeurosis is one important factor in predisposition to inguinal and femoral

hernia. They find that the transversus sheet, including the ligaments of Henle, Hesselbach, and Thomson is normally inserted into the ilio-pectineal line.

The type of repair advised by them therefore is on the lines of the Lotheissen operation for femoral hernia—the conjoined tendon being approximated behind the spermatic cord to the ilio-pectineal or Cooper's ligament. In order to avoid tension they also advise an incision into the internal oblique aponeurosis similar to that described by Tanner. At the same time it should be noted that Tanner's operation can be performed by substituting Cooper's ligament for the inguinal ligament, in the repair advised by him. This operation is further and more fully described under the treatment of femoral hernia.

Rectus Inguinal Herniorrhaphy. The posterior inguinal wall may be filled in and reinforced by a quadrilateral flap obtained from the internal oblique layer of the anterior rectus sheath. In Bloodgood's operation it is first necessary to separate the external oblique part of the sheath, as in the Tanner slide operation. The flap is cut in such a way that it hinges downwards round the fixed rectus sheath edge. It is sutured to Poupart's ligament.

DIRECT INGUINAL HERNIA

Definition. A protrusion of abdominal contents which reaches the inguinal canal through its posterior wall within the triangle of Hesselbach.

Classification. The hernia may remain confined to the inguinal canal but will then spread outwards through the external abdominal ring. It does not reach the scrotum, however. These herniæ commonly contain bladder within the wall of the sac or may be similarly related to the cæcum or pelvic colon.

Complications. Strangulation is rare. These herniæ are nearly always reducible and often bilateral. Symptoms may arise because of the presence of the bladder in the wall of the hernia.

Treatment. Since these patients are usually men of advanced years and as the herniæ only rarely strangulate, treatment by operation is often less strongly advocated. Many patients are treated satisfactorily by a truss. Chronic bronchitis, prostatic enlargement, and constipation will frequently require treatment. An elastic type of truss is a more reasonable and practicable alternative to a spring truss in this type of hernia than in the treatment of indirect inguinal hernia. Operative treatment is on the lines already discussed, and consists essentially in removal of the sac whenever possible by an approach as described under High Ligation Excision Herniotomy.

The sac of a direct hernia, however, does not lie within the spermatic cord but tends to lift it forwards as it reaches the inguinal canal from behind. It often has a broader neck than the indirect variety and frequently has the appearance of a dome-like bulge. These sacs may be associated also with either an indirect inguinal hernia or a femoral hernia. Care will be necessary in cleaning the sac because it is often thin and poorly defined, adherent to the bladder, and less clearly defined in relation to the surrounding anatomy. When the causative sac has been removed, the operation continues on the lines described under indirect inguinal hernia, using one of the methods of herniorrhaphy or hernioplasty. The Bassini operation, Bloodgood's and the Tanner slide operation and autogenous fascial hernioplasty by Gallie's method are in common use for this type of hernia.

SUPRAVESICAL HERNIA

Definition. Hernial protrusions which emerge in an area bounded below by the bladder, above by the umbilicus, and laterally by the obliterated hypogastric vessels. This is the area anatomically known as the internal inguinal triangle. Herniæ in this area are of rare occurrence.

Classification. The protrusion may pass near the apex of the bladder (apical) when it may be noted in conjunction with abnormalities of the urachus. More usually the hernia arises in a foveal depression in the serous covering of the bladder. From here it may burrow laterally and become a clinically external hernia as one of the varieties of direct inguinal hernia. This is a paravesical type. Very rarely the hernia may pass into the Cave of Retzius—a case having been described in which 8 ft. of bowel were found in a sac of this type (Hulke).

These supravesical herniæ usually manifest themselves as one of the forms of acute intestinal obstruction—the exact diagnosis being made following a laparotomy for this condition.

Treatment. This depends upon the variety of hernia and its method of presentation. In many cases a laparotomy for acute intestinal obstruction will alone show the position of the hernia, and treatment will then consist of reduction and closure of the sac, or removal when possible When this type of hernia appears externally in the inguinal region it will be necessary to remove the causative sac and to undertake a repair as in other forms of direct inguinal hernia.

FEMORAL HERNIA

Definition. A protrusion of abdominal contents along a sac which passes through the femoral ring, down the femoral canal and from there to the saphenous opening in the deep fascia of the thigh.

Classification. The sac and hernia may be internal or external It may be associated with an inguinal hernia and it may be irreducible or strangulated.

Diagnosis. In the classical type the hernia forms a swelling in the thigh below the line of the inguinal ligament This is usually reducible but often incompletely so, leaving a small hard lump at the saphenous opening. It occurs mostly in women who are obese and who have been pregnant, but it is also found in men and even in thin elderly spinsters. As with other hernial protrusions the swelling has an expansile impulse on coughing.

Differential Diagnosis. Indirect and direct inguinal hernia, a lipoma, inguinal adenitis, a saphenous varix, a psoas abscess, a Bartholin's gland swelling, and a femoral aneurysm must all be excluded.

Causation. This may be congenital Fauntleroy described a case in which the spermatic cord and an empty diverticulum of peritoneum passed through the femoral canal, the inguinal canal was empty and the testicle was found normally placed in the scrotum. Quilliam described another case in which the hernial sac passed through the internal abdominal ring and along the inguinal canal, but then passed through a *hiatus* in the inguinal ligament and thence to the femoral canal and fossa ovalis. Clinically this resembled a femoral hernia. These rare cases suggest that an abnormality of testicular descent may sometimes be the cause of femoral hernia Souttar, however, has produced evidence to suggest that the primary cause is an abnormality of the extra-peritoneal fat occupying the femoral opening and ring. The abnormally thick and fatty

covering over the sac of a femoral hernia is generally recognized. The possibility remains that the abnormal extraperitoneal fatty deposit may in some way belong to that rarely seen structure known as the gubernaculum.

Complications. *Intestinal obstruction due to internal hernia, irreducibility,* and strangulation commonly occur. The sac of the hernia is frequently related to the bladder wall and this may either cause urinary symptoms or post-operative complications due to injury to the bladder when freeing the sac.

Treatment by Truss. Whenever possible a truss should be avoided. Reduction is often incomplete and the hernia may under these circumstances produce symptoms of acute intestinal obstruction from a nipping of part of the wall of the small intestine in the upper part of the femoral canal (Richter's hernia). A femoral truss mechanically is unsound because the thigh is constantly moving; therefore such trusses, when they are essential, require a garter-like band to hold them in position round the thigh. This may cause pressure on the femoral or internal saphenous veins resulting in superficial or deep phlebitis. A spring truss which may be prescribed occasionally is built on similar lines to an inguinal truss, but it has a more elongated pad designed to reach the lower placed femoral opening. Very rarely an elastic truss may be supplied for this condition.

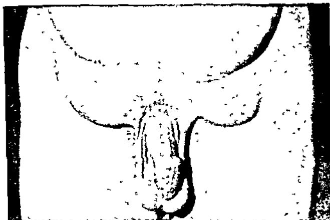
Operative Treatment. The femoral canal may be approached surgically either from above or from below the inguinal ligament. The inferior approach is less popular nowadays because the herniated viscera are not as adequately viewed as from the upper approach. The affected intestine after reduction may easily slide back into the peritoneal cavity and escape identification. At the same time surgical treatment after strangulation for the affected bowel either by the conservative application of hot packs or by resection is less adequately performed. In the rare cases when an abnormal obturator artery is present the control of bleeding from the lower approach (Lockwood's operation) is almost impossible.

When the canal is approached from below the inguinal ligament, a slanting incision is made over the most prominent part of the swelling in the thigh. Dissection will reveal the femoral sac to be somewhat like a lipoma with an attached pedicle emerging from the saphenous ring in close relation to the internal saphenous and femoral veins and the nearby femoral artery. The sac is always thick walled and after incising many layers the peritoneal sac will be found to be of relatively small size. The hernial contents are viewed and the hernia is reduced. When the omentum or bowel contained in the femoral sac is irreducible, it will be necessary first by gentle manipulation of the finger to see whether a little stretching of the canal and ring can be affected by the index finger. When this is not successful in bringing about reduction more easily, it will be necessary to cut either the thickened neck of the femoral sac or in some cases to cut into Gimbernat's ligament. It is at this stage that damage may be done to the abnormal obturator artery. After removal of the sac the repair of the hernial area is brought about by a series of stitches drawing the inguinal ligament to the pectineal fascia and closing the margins of the saphenous ring in the deep fascia.

When the upper approach is used, an incision is made as for inguinal hernia. The external oblique fascia is incised in the line of its fibres and the spermatic cord exposed in the inguinal canal. The cord or round ligament is then retracted upwards to expose the transversalis fascia in the posterior inguinal wall below the arching conjoined tendon. The fascia is picked up and a transverse incision made into it anterior anatomically to

the position of the femoral canal. The incision is deepened by blunt dissection and the position of the femoral vein and artery and also of the neck of the sac are confirmed as they pass beneath the inguinal ligament. At this stage, a small amount of retraction on the lower edge of the wound will allow the surgeon to attain good access to the sac of the hernia as it lies in the saphenous opening. With an adequate view of the femoral canal and hernia both from above and below the inguinal ligament it is now possible either by gentle stretching or by incision, as previously described, to obtain reduction of the hernia. The affected bowel is clearly seen and can be treated accordingly. It is at this stage that the specially designed hernia retractor is used to guide the surgeon's knife as he makes the necessary protected incision into the constricting ring.

The modern repair of the femoral hernial area necessitates an adequate view of the ilio-pectineal or Cooper's ligament, following removal of the sac and after having invaginated it upon itself from below upwards through the femoral ring under direct



(From "Hernia" by F Mitchell-Heggs, J & A Churchill Ltd, London)
FIG 170 Double femoral hernia.

vision. When doing this, it will be necessary to retract the femoral vein slightly in order to be able to insert the subsequent series of sutures into the ligament. The vein should never be retracted with metal retractors. Only the surgeon's index finger should be used for this purpose, so that no damage should occur to the vein either by retraction or by the suture needle. In fat patients the wound at this stage is often a deep one. Good light and hæmostasis are essential therefore, and the insertion of the sutures into Cooper's ligament will be made easier by either a handled needle or by a fish-hook type of needle on a long needle holder. Between two and five strong sutures preferably of unabsorbable suture material are inserted independently and the ends of the sutures clamped by hæmostats. The retraction is now removed and a repair performed by passing the individual sutures separately through the neighbouring edge of the conjoined tendon which is subsequently drawn as the ligatures are tied, down to Cooper's ligament. In order to avoid tension, an incision should be made into the internal oblique part of the anterior rectus sheath on the lines described under Tanner's operation for the repair of inguinal hernia.

Strangulated Hernia. The clinical picture of a strangulated hernia varies with its contents, the anatomical site of the protrusion, whether the hernia is an internal or an external one and the general health of the individual concerned. Internal herniæ which

covering over the sac of a femoral hernia is generally recognized. The possibility remains that the abnormal extraperitoneal fatty deposit may in some way belong to that rarely seen structure known as the gubernaculum.

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ox fascia, or may be a metallic or non-metallic substance. The substances chosen have been many and varied and each method has been found to have advantages and disadvantages. It may be said that the hernioplasty operations are *on trial*, and that their usefulness will not be fully proved until sufficient time has elapsed for both sides of the picture to have been ascertained. The operations are not undertaken in young adults or children, and are indicated only in the treatment of recurrent hernia, severe and longstanding hernia and in cases in which there is a loss of substance of the muscle or aponeurosis at the site of protrusion.

Hernioplasty	Autogenous	fascial sutures	pedicled	McArthur 1901
			free	Gallie and Le Mesurier, 1923
		fascial graft	pedicled	Wangensteen.
			free	Kirschner.
		muscle graft	rectus	Bloodgood.
			sartorius	De Goray.
		cutis graft		Mair 1940.
	Animal	ox fascia		Glasser. Maingot 1941.
		silk floss		Souttar 1922.
	Non-metallic	polythene		Thomson 1951.
		celluloid (obsolete)		
	Metallic	silver wire filigree		McGavin 1909.
		aluminium		Cole.
		tantalum mesh		Throckmorton.
		steel wire gauze.		Koontz 1948.

Autogenous (Thigh) Fascial Inguinal Hernioplasty. In this technique, which was popularized by Gallie, the weakened posterior wall of the inguinal canal is repaired by a darn of fascia lata obtained from the thigh. The strips may be obtained by making a long incision down the lateral aspect of the thigh and exposing the fascia lata to direct vision. They are cut some 6 in. or 7 in. in length and ¼ in. wide. The gap so made in the fascia lata is not sutured because it would be so under tension. The muscle hernia which results causes little or no disability. The skin and deep fascia are sutured in the ordinary way leaving a long linear scar which heals readily.

More commonly nowadays one of the forms of fascial stripper is used. In this method a small incision is made at the lower and outer aspect of the thigh and a small area of fascia is exposed and selected. A leaf of fascia lata is cut out to fit the fascial stripper knife and the operation is completed by pushing the introducer away from the skin wound. This strips the fascia to the required length and breadth. With certain strippers, a second incision is made in the outer part of the upper third of the thigh to free the fascial strip from the aponeurosis. In other varieties a special knife is included in the fascial stripper and by manipulating this the strip can be cut off with the instrument

are strangulated are as a rule only accurately diagnosed following a laparotomy performed for their relief. When an external hernia is present it will be irreducible and tender and these signs, together with those of acute intestinal obstruction which follow in the later stages, will make the diagnosis relatively simple. The treatment of strangulation in any hernia takes preference over any operative treatment designed to prevent a recurrence of the hernia. When a patient is very severely ill one should administer an intravenous saline drip transfusion, wash out the stomach and continue gastric aspiration during the operation with the aid of a long gastric tube. General anæsthesia should never be undertaken in this type of case without constant watch on the blood pressure and aspiration of the stomach. Unfortunately, owing to the nature of the strangulating condition, it is not possible to delay operation long; so that when operative time is short it may sometimes be necessary to reduce the strangulation and treat the affected gut only. A full hernial repair may then be undertaken later, when the patient has recovered from the effect of the initial strangulation.

When dealing with the sac of a strangulated hernia before it has been opened, the area should be well packed off with swabs which can be subsequently discarded. At the same time the theatre suction apparatus should be ready so that at the moment of incision of the sac the fluid contents can be quickly and cleanly removed. This fluid contains highly contaminated material which should not be allowed to enter the general peritoneal cavity or to reach the walls of the surgical incision. When the strangulated omentum, small or large intestine or other viscus has been freed from its hernial sac, it is carefully examined to discover whether there is a particularly tight constricting ring left on its surface. This mark is the danger point for a possible pressure gangrene which may result in a perforation of the affected viscus at a later date. Moderately hot moist saline packs are gently applied to the intestine and its colour, the shininess of its exterior, its odour, and the appearance or absence of peristalsis are particularly noted. Good prognostic signs are: a shiny peritoneal surface, a dark blue colour, the re-commencement of peristalsis and a minimal odour, whereas a dull surface with a black, green, or yellow colour, absent peristalsis and a fæcal odour are the reverse.

When resection of bowel is necessary this is performed in the usual manner. End-to-end anastomosis is probably the method of choice, the suture line consisting of two main layers. The outer line will include the peritoneal coat and the tissues immediately beneath it, while the inner suture line will include all layers of the intestine and is expected to be hæmostatic My own preference is for a fine unabsorbable suture material. The area of anastomosis following a resection of this type in strangulated hernia should be "drained" so that probable contamination may be anticipated by an exit route designed by the surgeon himself. Such a corrugated rubber "drain" can be removed after three days in most cases and should be regarded purely as an extra precautionary measure to prevent a subsequent abscess. The site should also be sprinkled lightly with one of the antibiotic powders such as Streptomycin or Penicillin.

Hernioplasty. This type of operation is undertaken when a permanent repair of a hernia is required but in which the previously described herniotomy and herniorrhaphy procedures are not considered adequate. It consists essentially of the repair of the hernial area concerned by the insertion, at the point of protrusion, of some other material than those at the site. The material used may be derived either from the patient's own soft tissues or from exogenous material which may be from a living structure, such as

described by Sampson Handley in 1918 but it was not until 1922 that Souttar introduced floss silk for this purpose. Floss silk is now supplied in ampoules each of which contains one yard of silk on a spool. It is made ready for use either by boiling it for about an hour, or by immersing in Bard Parker Antiseptic Solution for 30 minutes, then rinsing in sterile saline and later in doubly distilled water. One long length of floss silk is required for a repair.

The first anchoring part of this continuous stitch takes a firm bite of the periosteum over the pubic crest and the edge of the rectus sheath. The free end is not tied immediately, this being left until the darn has been completed and the return stitch has reached the point of commencement again. The suture continues laterally, without tension, forming a darn between the inguinal ligament and the conjoined tendon until the internal abdominal ring has been reached. It then returns medially taking its attachment from the inguinal ligament and conjoined tendon as before but *between* the stitches of the previous row. Maingot advises that small or medium size round bodied trocar pointed Mayo needles should be used.

Sepsis and hæmatoma formation must be avoided wherever possible. The subsequent wound breakdown may result in delayed healing with sinus formation and, in an intractable case, it may be necessary to perform a second operation to remove the floss silk. It has been objected that the spaces between the many fine strands of the silk may act as a good site for bacterial proliferation in a contaminated case. However, there are many who consider that there is a greater natural tissue reaction to sterile silk than there is to stainless steel wire, fascia, tantalum, cutis, or polythene.

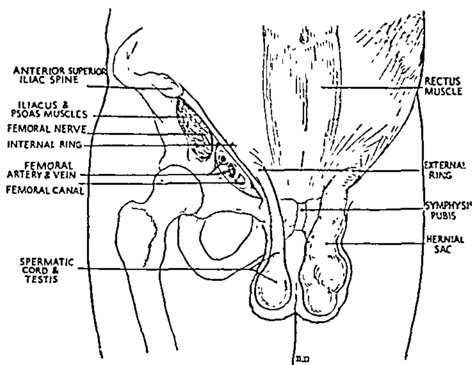
Tantalum Inguinal Hernioplasty. As an alternative form of hernioplasty, the posterior inguinal wall may be strengthened by the insertion of a mesh made of fine tantalum wire. Tantalum, as opposed to certain commonly and successfully employed substances used for internal splintage, is an element and not an alloy. It is obtained with difficulty from raw material which is itself rare and expensive The wire used is 0·003 in. in diameter, of high tensile strength, malleability, and ductility, and is woven into the form of a mesh 50 × 50 in size.

Tantalum is for practical purposes inert in the tissues both electrolytically and as regards its reaction to the surrounding tissues. The wire mesh is scrubbed with soap and water, rinsed, and sponged off with alcohol or ether. It is then sterilized by autoclave. The operation requires a standard inguinal approach, with removal of the sac and fascial repair when indicated The boundaries of the posterior inguinal wall or hernial defect are estimated and a section of the gauze mesh is cut by ordinary surgical scissors to fill the unretracted gap while allowing for a good margin around, and also sufficient mesh to include a turned over edge of 1 cm. breadth. The double thickness edge forms a firm non-tear medium for holding stitches which may be of tantalum wire or other unabsorbable suture material.

It has been found better to overestimate rather than to underestimate the size of the mesh used. It is fixed to the inguinal ligament, conjoined tendon, and rectus edge by sutures placed approximately 1 cm. apart. Tantalum gauze is an easy substance with which to work and will be found to lie well between the aponeurotic or other layers. No harm has been found to result from its near proximity either to a viscus, a nerve, or the peritoneum The repair anatomically is a modification of the Bassini method as the spermatic cord is placed at the end of the operation anterior to

in situ. When the latter variety of stripper is used only one small skin incision is therefore necessary.

When an adequate number of strips have been obtained, depending upon the size of the hernial defect, the first strip is threaded on to a Gallie needle. The posterior inguinal wall is then reinforced by making an interlacing to and fro darn with a number of strips to lie between the conjoined tendon and the inguinal ligament. No more tension is put on the fascial strips other than what is required to hold the darn securely in position. The purpose of the fascial darn is to act as a framework of living tissue upon which



(From "Hernia" by F Mitchell-Heggs, J & A. Churchill Ltd, London)

FIG 171 To show the relations of the femoral and inguinal canals to the pelvis A diagrammatic representation of a hernial sac containing omentum is given on the left side.

granulation tissue and fibroblasts can be expected in the course of time to form a firm fibrous barrier.

This form of hernioplasty has so far stood the test of time well. It is generally applicable and does not necessitate the insertion of any inert foreign body, apart from the fascia which is from the patient's own tissues. Recurrences quoted at between 15 per cent and 29 per cent may occur and the presence of a hæmatoma or sepsis may cause prolonged trouble. The necessity for using a large needle may cause tearing in the friable tissues or the complications of intractable leg pain and muscle hernia may arise. It will be realized also that the process of obtaining the necessary strips from the thigh, unless performed by an assistant, will prolong operation time. This type of hernioplasty may be regarded as a modification of the Bassini repair. The same principle can be used in the treatment of a severe femoral hernia, the fascial darn being made to lie across the opening of the femoral canal between the inguinal ligament and Cooper's ligament.

Inguinal Floss Silk Hernioplasty. The repair of difficult herniæ by a darn was first

causes are very likely to cause trouble when they occur, as in other forms of hernioplasty. Cases have also been described in which dermoid cyst formation has taken place following the insertion of a cutis graft.

Under normal conditions it has been found histologically that the cutis graft is gradually converted to fibrous tissue by a slow replacement of the epidermal and skin elements.

UMBILICAL HERNIA

Umbilical herniæ may be either congenital or acquired. The former type are seen usually in babies and young children, whereas the latter variety is para-umbilical in position and is found in adults, usually of obese build. The congenital infantile herniæ which pass through the centre of the umbilical scar are less likely to require operative treatment than those which emerge partly through the umbilical scar and partly from the tissues at its edge Strangulation is unusual but does occur in infantile cases and it is therefore wise to treat such herniæ in babies with some form of support following reduction. The simple and time honoured method uses a halfpenny piece which is fixed to the abdominal wall by a piece of strapping. As an alternative, an infant's rubber umbilical truss may be prescribed. Operation need not be undertaken until the child is about 5 months old and by this time it will usually be evident both to the parents and to the surgeon whether or not the hernia is of sufficiently small size to be likely to be healed by the appliance alone.



(From "Hernia" by F. Mitchell-Heggs. J. & A Churchill Ltd, London)

FIG. 172. Large infantile umbilical hernia in a child of 20 months

Infantile Umbilical Herniotomy. A curved incision approximately 2 in. in length is made to pass around the upper part of the circumference of the umbilicus The umbilical scar is dissected out and retracted downwards and during this dissection it will be found possible to demonstrate the position of the causative hernial sac. The skin flap is then pulled downwards leaving the sac of the umbilical hernia which is opened and inspected. Any hernial contents are reduced. The sac is removed and the small opening in the aponeurosis and peritoneum obliterated by one or two transversely placed sutures. The flap of skin which includes the natural umbilical scar is then replaced and the skin incision joined by several small and closely applied sutures. This operation leaves the child with a well concealed scar and a normal looking navel.

Mayo's Operation for Para-umbilical Hernia. Para-umbilical herniæ may be of small or large size and tend to occur in obese patients with a weak abdominal wall. They are frequently complicated by inflammation, irreducibility, and strangulation and these complications occur in patients who are generally a poor operative risk. It is better therefore that a patient suffering from this condition should have a radical operation performed as early as possible and when the hernia is small and uncomplicated.

An elliptical transverse skin incision is made to include the hernia within the area of the incision. This is deepened until the external oblique aponeurosis is identified. The wedge of tissue so formed will include the skin, subcutaneous tissue, and hernial

the tantalum gauze. The external oblique aponeurosis is sutured superficially to it in the usual manner.

Provided that sepsis and hæmatoma formation have been avoided, it is probable that a hernial repair performed with the aid of tantalum gauze will result in a cleanly healed wound. Occasionally a patient will be vaguely aware of the presence of the mesh if he knows of its existence and radiologically it will be seen that after 2 or 3 months the original mesh has become broken up and fragmented. The strength of the hernial repair is in no way affected by this because the fragments have been bound to the surrounding aponeurosis by a firm mass of fibrous tissue.

Tantalum gauze hernioplasty is also used in the repair of umbilical, diaphragmatic, and in particular, incisional hernia associated with tissue deficiency.

Stainless Steel Inguinal Hernioplasty. *An almost identical malleable wire mesh is* now available which is made of stainless steel wire as opposed to tantalum. Its indications and use are similar. The 50 × 50 mesh is made from 18–8 stainless steel monostrand wire of 0·003 in. diameter. This material is of course cheaper than tantalum and does not fragment when exposed to the bending strains which are found to occur in the inguinal region. This wire mesh may also be used in the repair of umbilical and incisional hernia.

Polythene Inguinal Hernioplasty. An alternative hernioplasty method includes the insertion of a polythene plate. This substance is easily obtained and inexpensive. It is made for surgical use in the form of a waxy-white thin flat plate which feels soapy to the touch. The material may be cut with scissors to any required shape and can be perforated with a Mayo needle. Sterilization is preferably by autoclave. The sheeting used is $\frac{1}{16}$ in. in thickness and an area is cut of somewhat oval shape, about $3\frac{1}{2}$ in. by $1\frac{1}{2}$ in. and including a small opening for the spermatic cord when necessary.

Autogenous Cutis Inguinal Hernioplasty. The posterior inguinal wall may also be strengthened by the insertion of a tense skin graft obtained from the patient's own tissues. The indications are as for other forms of hernioplasty. The skin incision is similar to the usual inguinal one but is elliptical and includes a strip of skin at least $2\frac{1}{2}$ in. long and 1 in. wide. The subsequent inguinal canal exposure with removal of the sac is on standard lines. The skin graft is obtained from the ellipse of the skin incision by freeing it from fat. The graft is then placed in saline and trimmed so that its parted ends are $\frac{1}{3}$ and $\frac{1}{4}$ in. in breadth. When it is ready for insertion into the posterior inguinal wall a double tail is made in the broader end by incising the graft in its long axis for $\frac{3}{4}$ in. and these tails are stitched to embrace the cord as it passes through the internal abdominal ring at the end of the operation.

Mair advises fixing the medial end of the graft to the lowest part of the inguinal ligament, the fascia over the symphysis and the anterior aspect of the rectus sheath by three sutures. The upper tails are next fixed above the level of the internal ring to the internal oblique expansion and to the shelving margin of Poupart's ligament above the ring. The lower and upper borders of the graft are sutured under tension to the inguinal ligament and to the aponeurotic area of the internal oblique very close to the lateral edge of the rectus sheath respectively. The skin graft should be tense and white.

The method does not make use of material other than the patient's own tissues. It has been objected, however, that the skin implant is of doubtful sterility and that a low grade pyrexia may result therefrom. Hæmatoma formation and wound sepsis from other

and practicable to tie off parts of the omentum which have become firmly fixed to t edges of the sac by adhesions.

After reduction of the hernial contents a large gap will be evident in both the pe toneum and the aponeurosis of the abdominal wall. It is usually possible by sha dissection around the margins of the umbilical ring to form a peritoneal layer and sew this up with interrupted sutures. The gap in the aponeurosis is then closed by formi

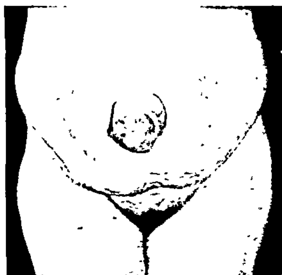


(a) (b)

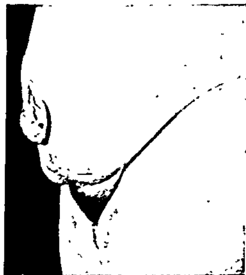
Fig. 174. A ventral hernia supported by an uplift type of surgical abdominal corset. (*From "Hernia" by F Mitchell-Heggs, J & A. Churchill Ltd, London*)

a flap in which the upper layer is brought downwards anterior to the lower layer. order to do this it is necessary to make a transversely placed incision on either side of umbilicus which extends to the edge of the rectus muscle. The sutures are insertec such a way as to draw the upper end of the lower flap upwards and underneath the up flap. They should be of strong and preferably unabsorbable suture material clo placed and reaching well to the edge of the defect which is a common site for recurre in this type of hernia. The repair of the aponeurosis is completed by a second rov stitches which fix the lower end of the upper flap to the lower part of the lower flap. skin of the abdominal wall of these patients is frequently of doubtful sterility and tl

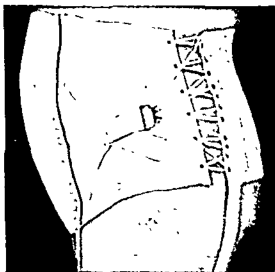
sac and the dissection proceeds until it is possible to lift away this tissue apart from the central area which is anchored by the hernial sac. The neck of the sac is then exposed by dissection and at a convenient point is grasped by two hæmostats placed close together. The tissue between these is incised and the hernial sac opened. It will be found that the



(a)



(b)



(c)

A para-umbilical hernia in an adult. (a) Anterior view, (b) lateral view, (c) supported by a surgical abdominal corset incorporating a circular, flat padded plate.

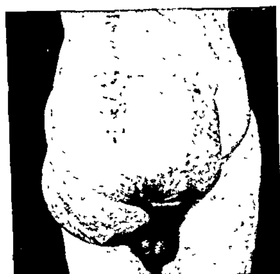
Fig. 173.

(From "Hernia" by F Mitchell-Heggs J & A Churchill Ltd, London)

contents of this type of hernia are commonly either the omentum or the transverse colon and that, whatever the viscus concerned, it is probable that there will be adhesions, particularly at the fundus of the umbilical hernial sac. It is for this reason that the neck of the sac is opened rather than the fundus The remaining margin of the neck of the sac is carefully cut through around the circumference, and the contents will then become visible, thus allowing their separation from it. In some cases it may be more convenient

treated in the early stages, when recurrence is detected, by a surgical abdominal corset.

Tantalum gauze has been successfully used to repair large congenital and acquired defects of the abdominal wall in which no other tissue lay between the peritoneum and



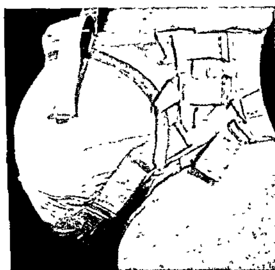
(a)



(b)



(c)



(d)

(From "Hern[illegible]

FIG. 175. Gross [illegible] the groin line with perineal st[illegible] ge pouch, lined chamois, to con[illegible] retain the belt in position and to spread the load.

the skin—both of which were buttonholed. Peritoneum may grow over and cover the mesh and granulation tissue will grow through it and allow the skin to epithelialize over its surface. At the same time infection of the wound does not necessarily prevent healing or result in sinus formation as is so frequent with silk or cotton sutures.

Concealed Abdominal Hernia. As far as retroperitoneal and intraperitoneal hernia are concerned there has been no marked change either in diagnosis or treatment except that in the improved treatment of acute intestinal obstruction. The use of pre-operative

may be intertrigo or frank sepsis in the skin in a case in which operation is immediately indicated owing to strangulation. It is therefore wise to complete the operation by a corrugated rubber drain which may be led out of the lateral edges of the wound.

Umbilical Hernioplasty. In this type of hernia when there is chronic bronchitis together perhaps with obesity, contamination of the wound and perhaps irreducibility or strangulation, recurrence of the hernia following operation may occur. When this happens a hernioplasty operation may be undertaken on the lines of that described for inguinal hernia and including the placing of a tantalum or steel gauze mesh, cutis graft, or polythene plate into the area of deficiency. The gauze mesh will be placed between the abdominal wall aponeurosis and the subcutaneous fatty layer. It should be of adequate size and fixed to an area 2 in. or more greater than the area of weakness.

Treatment by Surgical Support. It will frequently be found that operations for the treatment of para-umbilical hernia in adults are either inadvisable or that they cannot be undertaken for surgical or personal reasons. Provided that such herniæ can be reduced, a properly fitted surgical abdominal corset is a satisfactory and safe palliative measure. The corset should be of uplift type and individually made. It will probably be necessary to include steel or other supports for strengthening the back and it should, if possible, have a method of tightening or lacing which can easily be undertaken by these often obese and often old people. All such abdominal supports should be applied with the patient lying down and the hernia reduced. Incorporated anatomically over the umbilical region is a flat chamois covered metal pad.

When the hernia is irreducible but not strangulated, and operation is inadvisable, a specially made bag support of plastic material can be made to fit the hernial protrusion, when as much of it is reduced as possible, and this can be fitted to the corset around the umbilicus in order to prevent the hernia from becoming larger. It is of course only a palliative measure and does not prevent the dreaded onset of strangulation.

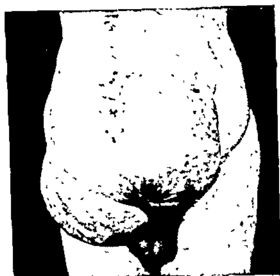
Incisional Hernia. Improved methods of anæsthesia, particularly in relation to muscle relaxation and the ability safely to anæsthetize a larger margin of patients, have enabled more of these cases to be treated surgically. They are often more extensive and time consuming than first appearances might suggest owing to multiple adhesions. Post-operatively, careful watching and treatment may be necessary because of abdominal distension or the more severe paralytic ileus.

The operation for incisional hernia is essentially a good and clean dissection of the abdominal wall and its layers. When there is a clear sac this should be removed. Re-apposition of the anatomical layers follows. The dissection requires skin towels and attention to hæmostasis. Asepsis should be meticulous. When there is tissue deficiency, or if the edges of the defect cannot be brought together without tension, the surgeon is well advised to use either tantalum gauze, a cutis graft, floss silk, or other accessory substance for repair. Many surgeons advise a fascial darn repair on the lines of the operation popularized by Gallie.

Unfortunately, particularly when the patient has a chronic cough, the recurrence rate in umbilical and incisional herniæ is higher than would be expected by those surgeons who do not have a long term follow-up clinic. The site of recurrence may be at either end of the hernial repair when a Mayo type of flap is used. Alternatively the abdominal wall is so weak that a general bulge ensues into which the omentum gradually slides. Such a recurrent hernia is an almost hopeless surgical risk and the patient will be better

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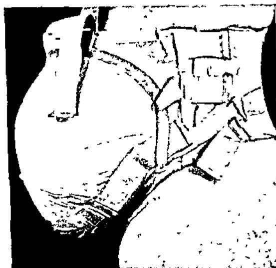
(a)



(b)



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(d)

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Fig 175 Gross [illegible] the groin line with perineal st[illegible] ge pouch, lined chamois, to cor[illegible] retain the belt in position and [illegible]

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stomach aspiration and the administration of intravenous fluids in a severely ill patient are of proved value. A straight X-ray of the abdomen, with the patient in a standing position if possible, will materially assist in the diagnosis of abdominal emergencies in which intestinal obstruction is suspected. The demonstration of fluid levels on the film, or the appearance known as the ladder pattern, are invaluable aids in early diagnosis. Post-operatively, fluid administration and gastric aspiration must now be regarded as standard modern treatment.

No particular advance has been made in the treatment of lumbar, epigastric, Spigelian and other forms of hernia apart from those coincident with the advance of surgery as a whole

DIAPHRAGMATIC HERNIA

Definition. A protrusion of abdominal contents through an opening in the diaphragm.

Causation. This condition may be congenital or acquired.

Classification. There may be a congenital failure in formation of the diaphragm in that one posterior half may be partially or completely absent; or there may be a failure of fusion between the anterolateral and central part formed from the septum transversum and the postero-lateral mesodermal remnant from the Wolffian body.

The pleuroperitoneal hiatus may persist as a complete opening with no pleura, no peritoneum and no sac, or there may be a small opening through the site of the foramen of Bochdalek with a pleural and peritoneal covering but no diaphragmatic representation. The hernia may pass through the subcostosternal foramen of Morgagni or the weakness may be in the region of the œsophageal hiatus or of the opening for the inferior vena cava.

Acquired diaphragmatic hernia may result from a wound, from a crushing injury, or because of disease, such as an empyema or subphrenic abscess, in a neighbouring organ.

Symptoms. When the disease is in its early stages, when no complications have developed, or if the hernia is very large, there may be no symptoms of any magnitude at all, and the diagnosis may be made during the course of a routine examination. Infants with this condition frequently die before the diagnosis is established, although survival to adult life may occur before symptoms arise.

More commonly, dyspnœa and cyanosis due to pressure on the heart and lungs occurs while the herniated stomach, small or large intestine, omentum or spleen may cause little in the way of symptoms apart from recurrent vomiting. In these infants, atelectasis is often diagnosed or the signs may suggest a pleural effusion, and aspiration without X-ray confirmation may result in injury to the herniated bowel. In other cases anæmia, recurrent vomiting, and loss of weight may resemble pyloric stenosis, or cyanosis might suggest a congenital heart lesion.

In adults, the symptoms produced are very varied, sometimes slight, sometimes severe, and often vague and misleading. This uncommon condition is classically a masquerader. Harrington found that cholecystitis, cholelithiasis, gastric ulcer, duodenal ulcer, hyperacidity, secondary anæmia, cardiac disease, carcinoma of the cardia, stricture of the œsophagus, appendicitis and intestinal obstruction, in that order of frequency were the previous erroneous diagnoses in his large series of cases.

Dyspnœa or cyanosis due to pressure on the heart or lungs, anæmia with loss of weight, melæna or hæmatemesis, obstructive symptoms due to twisting, volvulus or incarceration of one or other organ or heartburn, regurgitation and swallowing difficulties

due to œsophageal ulceration or inflammation, may be found. An adult patient is frequently a female and the indigestion picture is atypical and associated with changes in posture, particularly with stooping and with distension of the stomach. It may vary in intensity from severe spasms of pain suggestive of biliary colic to a vague indigestion in a pale and thin old lady who has an otherwise symptomless anæmia.

Diagnosis. This may be established following clinical examination, X-ray of the chest, a barium meal examination employing special methods or by œsophagoscopy. In many cases it is established at a laparotomy for the treatment of intestinal obstruction.

Pleuroperitoneal Hiatus Hernia. This hiatus is usually triangular with its apex towards the median part of the diaphragm. Such herniæ are very rare and are treated surgically by reduction followed by closure of the hiatus by sutures or by hernioplasty using either a thoracic or an abdominal approach. Frequently no hernial sac is present.

Subcostosternal Hernia. A very rare condition. Owing to the position of the hernia, its contents are more usually the cæcum or omentum than the stomach. A hernial sac is usually present, and the method of clinical presentation is either one of intestinal obstruction or of vague abdominal pain.

Posterior Diaphragmatic Deficiency Hernia. This is usually found in infants who are either very ill or dead. Only very rarely is the condition compatible with life to adult years. Treatment is surgical in the infant but in the adult will depend on the patient's symptoms, disability, age, and general condition.

Traumatic Diaphragmatic Hernia. This is one of the more common forms of diaphragmatic hernia. It results from wounding or from crushing injuries of the thorax or abdomen.

Post-inflammatory Diaphragmatic Hernia. This not uncommon form of hernia in the diaphragm follows an empyema, subphrenic abscess, liver abscess, or lung abscess. These result in necrosis and consequent weakening of the diaphragm thus predisposing it to herniation.

Vena Caval Hernia. Rarely a diaphragmatic hernia passes through a weak point in the region of this opening.

Congenital Short Œsophagus. In this rare condition, the cardia has always been placed above the level of the diaphragm, this being associated with a congenitally short œsophagus combined with a thoracic stomach. Macroscopically the site of junction between "stomach" forming the lower end of the gullet and the œsophagus is difficult to determine in these cases although microscopically or by the œsophagoscope this is more readily done. A barium swallow will usually show the position of the sphincter and materially assist in diagnosis.

This type is not suitable for surgery as the blood supply to the shortened œsophagus must not be disturbed in the process of freeing which is necessary in order to restore normal anatomy. Following the work of Allison, many cases which were formerly considered to be in this group are now classed as sliding hiatus herniæ, in which the shortening of the œsophagus is considered to be secondary to chronic inflammatory changes in the œsophageal wall

Œsophago-gastric Hernia. In this hernia the whole of the lower end of the œsophagus and part of the fundus of the stomach pass upwards through the œsophageal hiatus. The mediastinal"

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CHAPTER XIV

THE COLON

H. E. Lockhart-Mummery

CONGENITAL ABNORMALITIES

Faults in Rotation

These result both in abnormalities of position of parts of the colon, and usually also in deficient fixation. For a full account of the stages of intestinal development and rotation, and of the faults that may occur, the paper by Dott (1923) should be consulted.

Faults may occur both in the second and third stages of rotation.

In the Second Stage

(*a*) Non-rotation

The small intestine occupies the right side of the abdomen. The cæcum and ascending colon are in the midline, the transverse colon is very short, and the descending colon is in its normal position. The ileum enters the cæcum from the right side. The small intestine and ascending colon are attached only at the duodeno-colic isthmus, and a mid-gut volvulus is possible (volvulus neonatorum).

(*b*) Reversed Rotation

The transverse colon lies in a tunnel behind the duodenum and superior mesenteric vessels. Very few cases of this anomaly have been reported.

(*c*) Malrotation

The cæcum lies in the subpyloric region, and may be adherent to the liver and duodenum. As in non-rotation, mesenteric fixation is absent, and volvulus neonatorum can occur.

In the Third Stage. The cæcum may not be fully descended, or if descended is imperfectly fixed and may have a mesentery. The hepatic flexure and ascending colon are usually normal in position and fixation. This is the abnormality which may later lead to volvulus of the ileo-cæcal segment.

Atresia and Stenosis

These abnormalities are rare in the colon, accounting for only about 5 per cent of all intestinal atresias. When they do occur they may be multiple.

If an atresia or stenosis of the colon is found when operating for intestinal obstruction in a new born baby, an attempt at its relief by side-to-side anastomosis must be made. Ileostomy is poorly tolerated in new born babies. Mortality even with anastomosis is very high.

since the herniated stomach assists in the formation of the sac. This is the commonest form of diaphragmatic hernia. It has also been called the sliding hiatus hernia (Harrison) and the type 1 (Belsey).

Para-œsophageal Hiatus Hernia. The less frequent para-œsophageal type of hiatus hernia has been called the "rolling type" (Allison) and the type 2 (Belsey). In this variety there is a preformed sac lying in front of the œsophagus and this results in what Barrett has described as a Richter type of hernia. The fundus of the stomach lies within the sac, the lesser curvature remaining in its original position while the greater curvature and omentum are pulled upwards and become "upside down." The lower œsophagus in this type is in its normal position while part of the fundus herniates through the hiatus alongside the œsophagus. It is unusual in this type of hiatus hernia for "reflux" œsophagitis to occur although this condition is commonly found in the œsophago-gastric type.

Treatment. The treatment of diaphragmatic hernia depends on the age of the patient and the severity of the symptoms. Operation is usually undertaken in children once the diagnosis has been established and the child considered fit for operation.

In adults, medical treatment of hyperacidity and the adoption of suitable posture, on the lines of a duodenal ulcer regime in bed in hospital, is valuable in many cases particularly in hiatus hernia. Surgical intervention, either by the abdominal or by a thoracic route, consists in reduction of the hernia when possible and removal of an associated sac when present, together with repair either of the diaphragmatic aponeurosis or muscle.

When complications such as incarceration, volvulus, or acute intestinal obstruction develop, the treatment will primarily be aimed at the relief of these conditions by the most suitable surgical approach. Of herniæ of the diaphragm requiring surgery, Harrington places hiatus hernia first, those of disease or trauma second and congenital absence of part of the diaphragm, pleuro-peritoneal hiatus hernia and the subcostosternal varieties, in that order. The type of surgical intervention chosen will depend upon the state of the patient and the opinion of the surgeon concerned. Phrenic avulsion is often tried as a preliminary measure when œsophagoscopy and the use of bougies or other simpler methods of treating the constriction have failed. Following laparotomy, a partial gastrectomy or an œsophago-gastric resection with a jejunal anastomosis may be necessary in the treatment of hiatus hernia.

References

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Annals of Surg. 127, 1079.
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Gynae. Obst. 92, 419.

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References

[illegible] al Hernia, *Brit. J. Surg.* 29, 285.

[illegible] n Mesh in the Repair of Ventral Herniæ,

[illegible] *ae. Obst.* 88, 473

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Duplications

These are also rare anomalies, and may occur either as a double colon with two lumina in continuity, or as a cyst which does not communicate with the bowel lumen (enterogenous cyst). The duplication is always firmly united to the bowel, and the other characteristic features are a lining of intestinal epithelium and the presence in the cyst wall of a smooth muscle coat. (Evans, 1929.)

Symptoms may not occur for many years, sometimes not till adult life. The commonest presenting symptom is obstruction, but abdominal pain and even rectal bleeding have been recorded. A lump may be palpable on abdominal examination, but precise diagnosis is rarely possible. Treatment is by resection of the affected part of the colon; it is not possible to separate the bowel proper and the duplication by dissection, as the muscle coats blend.

MEGACOLON

THE term megacolon is used to cover those instances of colonic dilatation which occur in the absence of demonstrable organic obstruction, and are seen particularly in infancy and childhood. Only in recent years has it been appreciated that such cases fall into two groups which are clinically and pathologically distinct, and for which different treatment is needed (Bodian *et al.*, 1949). One group accord with the original description by Hirschsprung in 1887, while the other group do not show the same pathology, follow a more benign course, and respond more readily to treatment. Confusion between these two conditions has been largely responsible for the delay in solving the problem of megacolon in childhood.

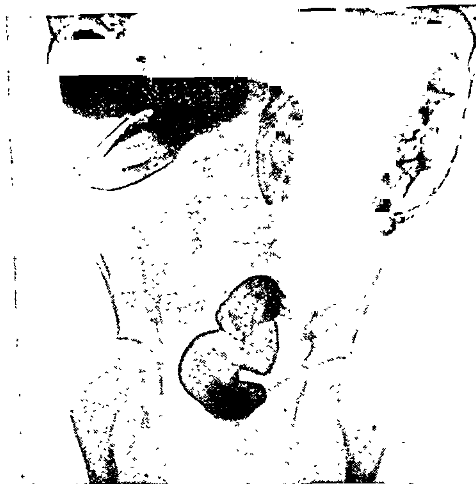
Hirschsprung's Disease

The disease is rare, being estimated to have an incidence of one case in 20–30 thousand births. Ninety per cent of cases occur in boys, and there appears to be some tendency for more than one member of a family to be affected, though the disease does not seem to be hereditary.

Pathology. The essential pathology of the disease is now known to be a congenital absence of parasympathetic ganglion cells from the terminal part of the large bowel. In 90 per cent of cases only the rectum and lower sigmoid colon are so affected, but rarely the affected segment may extend to the splenic flexure, cæcum, or even into the small bowel; the most extensive examples are probably incompatible with life. Owing to this deficiency the aganglionic segment cannot partake in co-ordinated propulsive movement, and thus tends to act as an incomplete obstruction to the colon. As a result the proximal colon dilates, often to an enormous size, and undergoes hypertrophy and secondary inflammatory changes. It is seen to become smaller fairly abruptly through a short "funnel" zone as it joins the narrow segment.

As a result of the great distension of the colon, the other viscera are compressed and the lower ribs are everted. Nutrition soon becomes deficient, and these children are usually undersized and underweight. Other serious congenital abnormalities are uncommon in association with megacolon. Bladder dysfunction is said to be frequently associated, but this is not the experience at Great Ormond Street, where bladder function in these children has been investigated and found to be normal (Williams, 1954).

Clinical Features. The patient is nearly always a boy, and there is a history of constipation from birth. There may even have been a delayed passage of meconium, and thereafter constipation of some degree has been constant. Distension is first seen at the age of a few months, varying in degree but never subsiding, and there may have been episodes of intestinal obstruction, relieved spontaneously or by enemata. The stools are



(Fanconi and Wallgren, "Textbook of Paediatrics." Heinemann)

FIG 176 A typical X-ray picture in Hirschsprung's disease

usually small and hard, but sometimes fluid fæces may be passed with much flatus, and with slight temporary diminution of distension. Pain is not a feature of the disease, either in the abdomen or on defæcation, and there is no fæcal incontinence.

On examination, the child is sallow, thin, and small for his age. The abdomen is greatly distended and tympanitic, with eversion of the umbilicus and flaring of the lower ribs. Fæcoliths are rarely palpable, but the outline of the distended colon may be made out. On rectal examination, the anus is seen to be normal, with no soiling. When a finger is passed, the rectum is found to be of normal calibre and empty; the loaded colon may sometimes be felt extra-rectally.

A barium enema will demonstrate the normal calibre of the rectum and recto-sigmoid, and show that the bowel above is greatly dilated and full of gas and fæcoliths. The examination must be skilfully carried out, as unless the filling is watched, the narrow part may so soon be obscured by the dilated bowel that its presence is not detected (Fig. 176).

Duplications

These are also rare anomalies, and may occur either as a double colon with two lumina *in continuity, or as a cyst which does not communicate with the bowel lumen (entero-*genous cyst). The duplication is always firmly united to the bowel, and the other characteristic features are a lining of intestinal epithelium and the presence in the cyst wall of a smooth muscle coat. (Evans, 1929.)

Symptoms may not occur for many years, sometimes not till adult life. The commonest presenting symptom is obstruction, but abdominal pain and even rectal bleeding have been recorded. A lump may be palpable on abdominal examination, but precise diagnosis is rarely possible. Treatment is by resection of the affected part of the colon; it is not possible to separate the bowel proper and the duplication by dissection, as the muscle coats blend.

MEGACOLON

THE term megacolon is used to cover those instances of colonic dilatation which occur in the absence of demonstrable organic obstruction, and are seen particularly in infancy and childhood. Only in recent years has it been appreciated that such cases fall into two groups which are clinically and pathologically distinct, and for which different treatment is needed (Bodian *et al.*, 1949). One group accord with the original description by Hirschsprung in 1887, while the other group do not show the same pathology, follow a more benign course, and respond more readily to treatment. Confusion between these two conditions has been largely responsible for the delay in solving the problem of megacolon in childhood

Hirschsprung's Disease

The disease is rare, being estimated to have an incidence of one case in 20–30 thousand births. Ninety per cent of cases occur in boys, and there appears to be some tendency for more than one member of a family to be affected, though the disease does not seem to be hereditary.

Pathology. The essential pathology of the disease is now known to be a congenital absence of parasympathetic ganglion cells from the terminal part of the large bowel. In 90 per cent of cases only the rectum and lower sigmoid colon are so affected, but rarely the affected segment may extend to the splenic flexure, cæcum, or even into the small bowel; the most extensive examples are probably incompatible with life. Owing to this deficiency the aganglionic segment cannot partake in co-ordinated propulsive movement, and thus tends to act as an incomplete obstruction to the colon. As a result the proximal colon dilates, often to an enormous size, and undergoes hypertrophy and secondary inflammatory changes. It is seen to become smaller fairly abruptly through a short "funnel" zone as it joins the narrow segment.

As a result of the great distension of the colon, the other viscera are compressed and the lower ribs are everted. Nutrition soon becomes deficient, and these children are usually undersized and underweight. Other serious congenital abnormalities are uncommon in association with megacolon Bladder dysfunction is said to be frequently associated, but this is not the experience at Great Ormond Street, where bladder function in these children has been investigated and found to be normal (Williams, 1954).

When first practised, it was usual to do this operation as part of a staged procedure, that is with preliminary transverse colostomy, followed by closure of the latter two or three weeks after the definitive operation. This not only entails three operations, but there is a tendency for a transverse colostomy in a baby to prolapse, adding further to the difficulties of management. Fortunately, increasing experience has shown that resection and anastomosis in one stage can be carried out with safety in most cases. Preparation must then be thorough, with mineral oil orally and low residue diet for some weeks, and repeated washouts in the last few days before operation. The latter should be given through a soft tube passed up into the dilated segment and fixed in position. (There is some danger of water intoxication in these patients if tap water is used for the washout, as absorption seems to be very active from the dilated colon. An isotonic solution should be used and the fluid run in carefully siphoned back.) In the event of acute obstruction supervening before operation, or in a child in very poor condition, a preliminary transverse colostomy is still certainly necessary and will result in complete subsidence of distension and great improvement in general health, allowing operation with reasonable safety after a few months.

Since 1948 this operation has been widely practised with consistently good results in all series published. Operative mortality is low (5 per cent or less) and all children who survive have been relieved of constipation and distension, with gratifying improvement in general health and gain in weight. No serious complications have been reported, and satisfactory rectal control has been achieved in all cases (Bodian *et al.*, 1951, Swenson, 1951). Relapse of the disease is rare, and is due to unintentional preservation of some aganglionic bowel. With greater experience these technical errors will cease to occur.

Functional Megacolon (Colonic Inertia)

As in true Hirschsprung's disease, this condition is commoner in boys. It is a form of constipation due to a combination of faulty training, bad habits, and sometimes a painful lesion in the anal canal. Considerable hypertrophy and dilatation of the rectum and recto-sigmoid may result due to the fæcal accumulation.

Clinically, there is usually a history of mild constipation from birth which becomes rather worse as the child grows. Large hard fæcal masses accumulate in the rectum, and may be felt on abdominal examination. These masses are painful for the child to pass, so a fear of defæcation is developed which makes the matter worse; the fæcal obstruction may result in an overflow diarrhœa with incontinence, while administration of purges causes colic and aggravates the diarrhœa without emptying the rectum.

Both rectal examination and X-rays confirm the presence of a dilated rectum, which may be packed with fæces; there is no narrow segment as in Hirschsprung's disease. The anal canal is usually normal though there is often soiling of the skin. The fæcal impaction is the only obstruction.

Treatment. This consists in the evacuation of the bowel and the re-training of the child in good habits. Treatment should be started under supervision in hospital, and the first essential is to empty the rectum by digital removal or washouts, if necessary under anæsthesia. The child is then started on a good mixed diet and bland aperient, and daily washouts are given for the first week. Thereafter natural defæcation should continue to occur, but regular habits must be enforced, and a washout given if the bowel does not move spontaneously. The child may be allowed home after a few weeks, but

Treatment. Untreated, the disease is fatal in about 30 per cent of cases before the third year of life, most of the deaths being from intestinal obstruction. The survivors continue with the disease with chronic ill-health, abdominal enlargement, and still liable to attacks of obstruction, and there is no tendency to spontaneous regression. Treatment by washouts and by operations on the sympathetic system have no lasting effect in the course of the disease, and "cures" in the past claimed for these methods were almost certainly not on cases of true Hirschsprung's disease. Colostomy results in subsidence of distension within a few months, and leads to great and lasting improvement in general health, but surgeons have naturally been reluctant to inflict the disabilities of a colostomy on a young child.

When it was appreciated that the distal segment was the abnormal part and was acting as an obstruction, it became possible to plan a rational treatment for the disease.

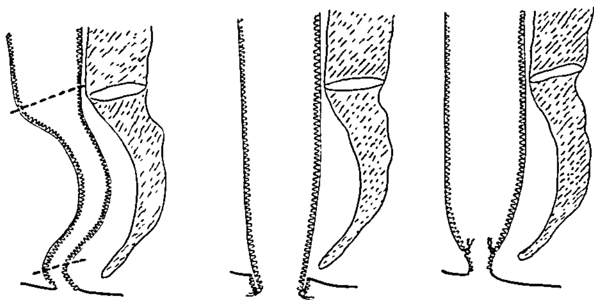


FIG 177. Diagram to show the extent of resection necessary in the operative treatment of Hirschsprung's disease, and the method of anastomosis

It is necessary to remove the aganglionic segment and to restore continuity, and this can be achieved by resecting the rectum and lower sigmoid, with anastomosis of normal colon to the anal canal. Such a low anastomosis requires both an abdominal and perineal approach, and the techniques now used are modifications of a method first described by Maunsell in 1892 and Weir in 1901. Great credit is due to Swenson (1948) in the United States who first applied the principles of the method to this disease.

The operation (often called Rectosigmoidectomy) is best carried out in infancy but after the child is weaned. After opening the abdomen, the rectum and recto-sigmoid are freed down to the pelvic floor, and it is important to keep this dissection close to the rectum so as not to damage the pelvic nerves and interfere with bladder function. The diseased segment is then resected and continuity restored by everting the anal canal, drawing the normal colon through it, and there performing an anastomosis which is returned inside the anus on completion (Fig. 177). Details of technique vary in the method of Swenson (1950) and that of Denis Browne (1949) and Stephens at Great Ormond Street, and for these the original papers should be consulted.



(Mr. O V. Lloyd-Davies' case)

Fig 178 Plain X-ray of the abdomen in a case of volvulus of the sigmoid colon The enormous distension of the single loop is characteristic.

should be regularly and frequently examined to ensure that fæcal impaction does not recur undetected. After some months when normal habits are again established the dose of aperient may be reduced, and perhaps eventually discontinued.

The syndrome is more common than Hirschsprung's disease, and gives rise to no mortality though it causes much distress. Treatment along the lines indicated may need to be prolonged, but a good result can finally be anticipated in most cases. Rarely, intractable constipation may persist into adult life, with great distension of the colon. Extensive resections of the colon often produce considerable improvement in such cases.

VOLVULUS

A VOLVULUS occurs when a loop of bowel becomes twisted on itself around the axis of its own mesentery. Though volvulus of the transverse colon has been described, it is extremely rare because of the extensive attachment of the transverse mesocolon, whereas volvulus of the sigmoid colon and of the cæcum are seen more frequently.

Volvulus of the Sigmoid Colon

Incidence and Ætiology. Sigmoid volvulus is a comparatively rare cause of intestinal obstruction in this country, probably accounting only for 2–4 per cent of all cases. In other parts of the world it appears to be more common, and in Eastern Europe and Russia it is said to be the cause of a third of all cases of intestinal obstruction.

The condition predisposing to volvulus is the presence of a long and freely mobile loop of intestine with a narrow mesenteric attachment. A normal sigmoid loop partly fills these conditions, and the following additional factors may play a part.

(1) A congenitally long sigmoid loop—said to be commoner in men.

(2) Chronic constipation—resulting in loading and perhaps further elongation of the loop.

(3) Fibrosis in the base of the mesentery—sometimes seen in elderly people, and drawing the two ends of the loop towards each other.

(4) Changes in intra-peritoneal relations, as seen during pregnancy and in the puerperium.

Once the main factors exist, the actual inciting cause may then be trivial, such as muscular effort, coughing, purgation or a dietary indiscretion.

It is probable that the variation in incidence of this condition throughout the world is due largely to the difference in diet and in bowel habit of the various peoples. Similarly its comparative prevalence in mental hospitals is probably due to the faulty habits and constipation which often characterize these patients.

Pathology. A volvulus of the sigmoid colon produces three pathological changes.

(1) A "closed-loop" obstruction in the affected segment (*see* p. 409).

This leads to great gaseous distension of the loop which may reach enormous dimensions, sometimes filling the abdominal cavity. The high pressure within the lumen diminishes blood flow and thus impairs the nutrition of the bowel wall.

(2) Obstruction of the vessels in the root of the mesentery.

If the twist is tight, complete venous and arterial obstruction may be caused, which will rapidly lead to gangrene of the bowel. If the vascular obstruction is incomplete, the



(*Mr O V Lloyd-Davies' case*)

FIG. 178 Plain X-ray of the abdomen in a case of volvulus of the sigmoid colon. The enormous distension of the single loop is characteristic.

partial venous obstruction causes engorgement and œdema of the bowel wall and further impairs its nutrition, already jeopardized by the high intra-luminal pressure. A blood-stained effusion is poured into the peritoneal cavity and into the lumen of the affected loop.

(3) A simple mechanical obstruction of the bowel above the affected loop.

Distension of the rest of the colon results, but this is seldom marked nor has it any serious consequence in the early stages, when the closed-loop obstruction dominates the picture. In cases where the twist is only moderately tight, a ball-valve effect may be produced, so that gas may enter the loop from the proximal colon, but cannot escape. These "sub-acute" cases, which are relatively common, often show the greatest gaseous distension of the loop, and only partial obstruction of the proximal colon.

The twist may be either clock-wise or anti-clockwise and is commonly of 180 degrees, but two or even three complete turns are occasionally met with.

Clinical Features. Men are more frequently affected than women, and most patients are elderly. The onset is usually sudden, with severe and constant lower abdominal pain, to which spasms of colic may be added. Constipation is absolute, neither flatus or fæces being passed, though in cases where the twist is tight the patient may complain of tenesmus and pass a little bloody mucus. Distension occurs early and progresses rapidly, and is always a striking part of the clinical picture; it may become extreme within a few hours, and sometimes leads to respiratory embarrassment. Loud borborygmi may be audible. Vomiting is unusual until a late stage.

On examination, the general condition is usually well maintained, though in cases where there has been extensive infarction of the bowel there is considerable shock. The abdomen is distended and tympanitic in all cases, sometimes to such a degree that no other features can be made out on examination; in cases seen earlier the outline of the distended loop may be felt and is often tender. On rectal examination, the rectum is empty, and a little bloody mucus may be found on the examining finger.

Diagnosis. The most characteristic feature of a volvulus is the degree of distension and the rapid rate at which it occurs. The most useful aid to diagnosis is a plain X-ray of the abdomen: the typical picture of a sigmoid volvulus is of a single enormous loop outlined by gas, and containing a variable amount of fluid (Fig. 178). It must be remembered that the volvulus has also caused a simple mechanical obstruction of the colon; the cæcum and proximal colon may therefore be distended also, and this may lead to mistaken diagnosis unless the film is carefully studied.

When symptoms and signs are classical, correct diagnosis is usually possible. The main differential diagnosis is from other forms of large bowel obstruction, particularly those of several days duration where the distension may be considerable.

Treatment. Before any specific treatment for the volvulus is undertaken, it is important to overcome shock (by blood-transfusion if necessary), to correct dehydration, and to prevent vomiting and gastric distension by the passage of a Ryle's tube.

Though operation may be necessary, it is often worth trying to pass a long rectal tube or soft stomach tube through a sigmoidoscope into the twisted loop. This has the advantage that it can be attempted in the patient's bed with little disturbance and that if successful the loop is deflated and spontaneous untwisting nearly always occurs, thus effectively overcoming the obstruction (Bruusgaard, 1947). The tube should be passed well up into the loop and fixed to the perianal skin, and should be retained for two or

three days or until the distension is fully relieved. It is important to realize that the factors that led to the volvulus are still present, and that recurrence is likely. The patient should be advised to undergo laparotomy after suitable bowel preparation, when the offending loop may be safely resected.

There is no danger to the patient in a trial of this method, provided that manipulations are gentle and not persisted in too long. Patients in good general condition, and particularly those with a history of previous similar attacks which have relieved spontaneously, are particularly suitable for a trial of intubation, as such patients are unlikely to have a tight twist with circulatory interference. The possible danger of failing to operate for non-viable bowel can hardly occur in practice, as in such cases the tube will not pass. Nevertheless, laparotomy must be undertaken unless satisfactory clinical resolution of symptoms and signs is obtained, and in any case in which strangulation is suspected.

In those cases in which intubation is not indicated or has failed, urgent laparotomy is necessary. A long paramedian incision is made, so that the anatomy and pathology can be quickly determined. If the loop is viable, it should be untwisted and a long rectal tube passed by an assistant and guided by the surgeon's hand into the loop, which is thus deflated. Sometimes the distension is so extreme that the base of the loop cannot be made out: in such cases it should be punctured with a needle and deflated by suction, when it can usually be untwisted and a tube passed as before. The further treatment of the deflated but still enlarged bowel must vary with the particular circumstances. Fixation of the loop in the left iliac fossa with sutures or by tube drainage through a stab incision is sometimes of value, but is not always successful in preventing recurrence; if symptoms suggestive of recurrence occur later, the patient should be most strongly advised to submit to laparotomy and resection. If the loop is very large and the patient's condition permits, immediate resection by the Paul-Mickulicz method may be the wisest course.

In those cases when the bowel is not viable, resection is necessary. In most cases a Paul-Mickulicz operation with exteriorization of both ends can be rapidly accomplished, but sometimes the gangrene may extend so far distally that viable bowel cannot be brought to the surface. In such circumstances, the gangrenous bowel must be resected, the distal end of colon oversewn, and the proximal end brought out as a colostomy (Hartmann's operation). Continuity can usually be restored at a later date.

The prognosis will naturally depend on the age and condition of the patient, the condition of the bowel and the operation necessary. Successful intubation should result in a negligible mortality, whereas if resection is necessary mortality may well be 50 per cent or more.

Volvulus of the Cæcum

Incidence and Ætiology. This is a very rare condition, which should really be called "volvulus of the ileo-cæcal segment," as the lowest few inches of ileum and part of the ascending colon always twist with the cæcum. Such a volvulus can only occur when the cæcum and ascending colon are not properly fixed in their retro-peritoneal position, that is when there has been a failure of the third stage of rotation. Such a congenital failure of fusion is always found in these cases, and in addition there appears in many cases to have been some narrowing at the base of the mesenteric attachment (Lyall, 1946).

Pathology. The same pathological changes occur as are seen in a sigmoid volvulus.

partial venous obstruction causes engorgement and œdema of the bowel wall and further impairs its nutrition, already jeopardized by the high intra-luminal pressure. A blood-stained effusion is poured into the peritoneal cavity and into the lumen of the affected loop.

(3) A simple mechanical obstruction of the bowel above the affected loop.

Distension of the rest of the colon results, but this is seldom marked nor has it any serious consequence in the early stages, when the closed-loop obstruction dominates the picture. In cases where the twist is only moderately tight, a ball-valve effect may be produced, so that gas may enter the loop from the proximal colon, but cannot escape. These "sub-acute" cases, which are relatively common, often show the greatest gaseous distension of the loop, and only partial obstruction of the proximal colon.

The twist may be either clock-wise or anti-clockwise and is commonly of 180 degrees, but two or even three complete turns are occasionally met with.

Clinical Features. Men are more frequently affected than women, and most patients are elderly. The onset is usually sudden, with severe and constant lower abdominal pain, to which spasms of colic may be added. Constipation is absolute, neither flatus or fæces being passed, though in cases where the twist is tight the patient may complain of tenesmus and pass a little bloody mucus. Distension occurs early and progresses rapidly, and is always a striking part of the clinical picture; it may become extreme within a few hours, and sometimes leads to respiratory embarrassment. Loud borborygmi may be audible. Vomiting is unusual until a late stage.

On examination, the general condition is usually well maintained, though in cases where there has been extensive infarction of the bowel there is considerable shock. The abdomen is distended and tympanitic in all cases, sometimes to such a degree that no other features can be made out on examination; in cases seen earlier the outline of the distended loop may be felt and is often tender. On rectal examination, the rectum is empty, and a little bloody mucus may be found on the examining finger.

Diagnosis. The most characteristic feature of a volvulus is the degree of distension and the rapid rate at which it occurs. The most useful aid to diagnosis is a plain X-ray of the abdomen: the typical picture of a sigmoid volvulus is of a single enormous loop outlined by gas, and containing a variable amount of fluid (Fig. 178). It must be remembered that the volvulus has also caused a simple mechanical obstruction of the colon; the cæcum and proximal colon may therefore be distended also, and this may lead to mistaken diagnosis unless the film is carefully studied.

When symptoms and signs are classical, correct diagnosis is usually possible. The main differential diagnosis is from other forms of large bowel obstruction, particularly those of several days duration where the distension may be considerable.

Treatment. Before any specific treatment for the volvulus is undertaken, it is important to overcome shock (by blood-transfusion if necessary), to correct dehydration, and to prevent vomiting and gastric distension by the passage of a Ryle's tube.

Though operation may be necessary, it is often worth trying to pass a long rectal tube or soft stomach tube through a sigmoidoscope into the twisted loop. This has the advantage that it can be attempted in the patient's bed with little disturbance and that if successful the loop is deflated and spontaneous untwisting nearly always occurs, thus effectively overcoming the obstruction (Bruusgaard, 1947). The tube should be passed well up into the loop and fixed to the perianal skin, and should be retained for two or

widespread ulceration. While this description of two forms of the disease is largely true and convenient for descriptive purposes, it must be realized that they are not separate entities; ulcerated and hyperplastic areas may be seen together in one patient, and a careful search for other foci should always be made before regarding a lesion as solitary. Both types are now becoming rarer, due to the better control and treatment of pulmonary tuberculosis, and to the better control of milk supplies.

Pathology

The route of infection in all cases is enterogenous, due to the swallowing of infected material (milk or sputum). In most cases the first lesion occurs in the ileo-cæcal region, because this is an area with abundant lymphoid tissue, and perhaps because there is normally some intestinal stasis in this region. From this site there may be spread of the disease proximally into the small intestine or distally into the colon, and the regional lymph-nodes are early and constantly involved. If resistance is high the lesion in the bowel may heal leaving no trace, and the affected lymph-nodes calcify—this is the usual course in children, and accounts for the common finding in healthy adults of calcified mesenteric nodes with an apparently normal bowel.

If resistance is low or large numbers of the organism are swallowed, extensive ulceration occurs with spread to other areas of bowel (Ulcerative Form). The ulcers are characteristically shallow and undermined, tend to encircle the bowel, and on microscopic examination have the typical histology of tuberculosis. The disease spreads through all the coats of the bowel and involves the serosa, but the process is in most cases sufficiently slow to allow adhesions to form, and perforation into the general peritoneal cavity is uncommon. Such lesions may be widespread throughout most of the small intestine and colon, usually with areas of normal bowel between the diseased segments, but are more numerous near the ileo-cæcal valve. The encircling nature of the ulcers is likely to lead to some degree of stenosis in the small bowel, particularly as healing takes place, and obstruction may result. However, obstruction occurring during or after intestinal tuberculosis is more commonly due to adhesions and kinking of bowel from the serosal involvement than to stenosis of the lumen.

If resistance is higher, a more chronic form of the disease is seen, in which a localized lesion forms with great thickening of the bowel wall (Hyperplastic Form). Most usually found in the ileo-cæcal region, it may rarely affect other parts of the small bowel and colon. Macroscopically the affected bowel is thick and rigid and may be shortened, and in its commonest situation the cæcum and terminal ileum are drawn up towards the liver. Tubercles are sometimes seen on the serosal surface, and the lesion is usually sharply localized Regional lymph-nodes may be enlarged. On opening the bowel, the lumen is narrowed, the mucosa is rough and nodular, and there may be some ulceration, though the latter is not a marked feature. Microscopically, the main changes are œdema of the submucous and subserous coats, with infiltration of lymphocytes and endothelioid cells. Giant cells may be seen, but caseation is uncommon, and it is rare to be able to demonstrate the bacilli, either by staining or by animal inoculation. It will be appreciated that the histology is very similar to that of Crohn's disease, and indeed microscopic differentiation of the two conditions may be impossible. For certain diagnosis of tuberculosis, either the bacilli must be demonstrated, or typical tubercles with caseation must be found in bowel or lymph-nodes.

Thus a simple mechanical obstruction of the lower ileum is produced, and a closed-loop obstruction of the involved segment, with vascular occlusion of varying degree in the latter.

The twist is in a clockwise direction, and is usually only of 180 degrees, though tighter twists have been reported. It is rare for the vascular obstruction to be sufficient to cause early gangrene, but the cæcal distension is rapid, and this combined with the partial vascular obstruction may lead to perforation of the thin wall within a few hours.

Clinical Features. The condition tends to affect a younger age-group than does volvulus of the sigmoid, and most patients seen are between 20 and 40 years old. There is abdominal pain, central at first and later settling in the right lower quadrant, to which spasms of colic may be added. Constipation is usually absolute, but there may be one bowel action as the colon empties. Vomiting occurs soon after the onset of pain, and may be repeated and distressing. On examination, the general condition is well maintained until perforation occurs. There may be some generalized abdominal distension, and the outline of the distended cæcum can usually be made out as an ill-defined tender tympanitic swelling, more often in the central abdomen than in the right iliac fossa. In later cases, the characteristic ladder pattern of ileal obstruction may be seen, and the cæcal distension will be more prominent and more tense.

The most valuable aid to diagnosis is again the plain X-ray of the abdomen, best taken with the patient supine. This will show the distended coils of lower ileum and the gaseous distension of the cæcum, and should suggest the correct diagnosis. The condition most likely to mislead the clinician is a simple large bowel obstruction, particularly an obstruction in the right colon where cæcal distension is early and rapid.

Treatment. Early laparotomy is essential. The passage of a small tube for gastric aspiration, and the correction of dehydration by intravenous fluids are necessary preliminaries, but no time should be wasted in attempting to pass an intestinal tube. On opening the abdomen the distended cæcum will present and may require puncture and aspiration before it becomes possible to see and deal with the twist. The bowel should be untwisted, and further steps will depend on its viability. If obviously viable, it is as well to attempt fixation, and this may be done either by suturing to the parietal peritoneum of the flank, or by carrying out a cæcostomy, which can be allowed to close after a few weeks. Drainage by cæcostomy tube is probably wiser if the cæcum has been very distended and congested, but neither method is a certain guarantee against recurrence.

If the bowel is gangrenous, resection will be necessary. A right hemi-colectomy with anastomosis of ileum to transverse colon is the ideal if the patient's condition permits. Exteriorization procedures in the right colon are best avoided, but if the patient's condition is desperate and the operation must be terminated rapidly, the ileum and transverse colon may be brought out of the abdomen, and a Paul's tube tied in each end. Later closure can be undertaken after careful preparation.

TUBERCULOSIS

INTESTINAL tuberculosis may occur as the sole manifestation of tuberculous infection, or as a complication of pulmonary disease. In the former type a localized hyperplastic lesion is usually produced, while when secondary to lung disease the lesion is more often one of

tuberculosis or with any other active focus, a course of medical treatment should follow the operation. Patients are usually restored to normal health and recurrence of disease in the bowel is rare.

Sometimes the presence of some degree of intestinal obstruction may make resection and anastomosis in one stage hazardous, and ileo-transverse anastomosis should then be done as a first stage. Often such an anastomosis will lead to complete relief of symptoms, but even so it is usually preferable to remove the diseased bowel by a second operation, performed when the obstruction has been overcome and the patient in good condition. It should be remembered that the hyperplastic form of the disease is sometimes met with as part of a more widespread involvement of the intestine. Search of the remainder of the bowel is therefore advisable in every case, before the nature and extent of the operation is decided.

AMŒBIASIS

THE surgeon who works in an area where amœbic dysentery is endemic will soon become familiar with the manifestations of the disease. However, sporadic cases of amœbic infection may be seen in temperate zones, particularly when so many men and women served during the recent war in tropical areas, and it is therefore important that surgeons should be familiar with certain aspects of the disease. A definite history of dysenteric infection is not always obtained, and rarely an affected patient will be seen who has never been abroad.

In acute cases, free perforation of an ulcer in the colon or a walled-off perforation forming a peri-colic abscess may sometimes require surgery. The diagnosis in these cases is seldom in doubt. More confusing is the patient with an amœbic infection of the cæcal region, who may present with great clinical resemblance to appendicitis, often without diarrhœa. Appendicectomy is dangerous, and in endemic areas the stools should be examined for amœbæ before operating for appendicitis, and anti-amœbic drugs given if there is any past history of amœbiasis. If the correct diagnosis is not made until the abdomen has been opened, it should be closed again without removing the appendix.

Chronic amœbic infection of the colon seldom results in a diffuse colitis, but tends to involve particularly the cæcal and recto-sigmoid regions. Sometimes a granuloma (amœboma) will form and lead to a palpable mass, but frequently the infection leads only to a fairly localized granulomatous thickening of bowel wall, which can only be detected by rigidity or mucosal changes on X-ray examination. In either case diagnosis may be difficult, as bowel symptoms are seldom marked, and the amœbæ may be found in the stools only after prolonged and repeated search. A normal rectal mucosa on sigmoidoscopy does not exclude the presence of amœbic infection more proximally.

Diagnosis can usually be established if the possibility is thought of, and as the condition clears up rapidly on anti-amœbic drugs, a therapeutic test may be of value if there is doubt. A very small percentage of cases of amœbic dysentery may be followed, perhaps after several years of normal health, by chronic ulcerative colitis. This may also occur following bacillary dysentery, but it does not follow that ulcerative colitis, even in these cases, is due to chronic dysenteric infection.

Clinical Features

Ulcerative Form. This is most common between the ages of 20 and 40, being seen more often by the chest physician than by the surgeon in hospital practice. The main symptoms are abdominal pain, change of bowel habit, and loss of weight, but any of these symptoms in a sanatorium patient who is not progressing as expected may lead the physician to suspect intestinal disease. The main importance of this form of the disease to the surgeon is to emphasize the necessity of a chest X-ray in the investigation of abdominal disease.

Hyperplastic Form. In most reported series, the average age of onset of this form of the disease is 30 years, but cases may be seen from the ages of 16 to 60 or over. The symptoms are similar to those of the ulcerative form, but are of more insidious onset and of longer duration. Abdominal pain is the most common symptom: both lower abdominal aching and attacks of colic may occur. Bowel habit is altered, often with diarrhœa, but sometimes bouts of constipation are frequent and may culminate in intestinal obstruction. Some loss of weight is usual. Fever, and bleeding from the bowel are rare symptoms. Examination will usually reveal a palpable elongated tumour in the right iliac fossa.

The symptoms and signs are very similar to those of carcinoma of the cæcum and Crohn's disease, while sometimes other causes of a mass in the right iliac fossa, such as actinomycosis, amœbic granuloma, and appendix abscess may need to be considered in diagnosis. The diagnosis must rest largely on the history and physical signs, but X-rays both of the chest and the bowel are necessary. The barium enema will show a long filling-defect in the involved part, and will often demonstrate the shortening of the cæcum and ascending colon when that area is diseased. Barium meal is of greater value in detecting involvement of the small bowel. Examination of the stools for acid fast bacilli is of little value—they are rarely if ever found in the hyperplastic form of the disease, and are no indication of intestinal disease if found in the presence of open pulmonary tuberculosis. A negative Mantoux test is valuable in excluding tuberculosis, but a positive test is of little value.

Even at laparotomy, certain diagnosis may be impossible unless tubercles are visible. Patients with hyperplastic tuberculosis sometimes undergo laparotomy and resection with a diagnosis of carcinoma, the pathological report coming as a welcome surprise to the surgeon.

Treatment

Ulcerative Form. In most cases this occurs as part of widespread disease affecting both the respiratory and intestinal tracts, and no surgical intervention is indicated unless obstruction should supervene.

The prognosis of such cases has been enormously improved by modern drugs, and in the event of laparotomy being undertaken before the diagnosis becomes apparent, it is wiser to close the abdomen and institute chemotherapy. If a localized lesion or group of lesions is found, resection is probably beneficial, but should be followed by a full course of chemotherapy.

Hyperplastic Form. Resection with anastomosis is the treatment of choice in this form of the disease, whether it occurs in the ileo-cæcal region or in other parts of the colon. The prognosis after resection is good, but in patients with a history of active

tuberculosis or with any other active focus, a course of medical treatment should follow the operation. Patients are usually restored to normal health and recurrence of disease in the bowel is rare.

Sometimes the presence of some degree of intestinal obstruction may make resection and anastomosis in one stage hazardous, and ileo-transverse anastomosis should then be done as a first stage. Often such an anastomosis will lead to complete relief of symptoms, but even so it is usually preferable to remove the diseased bowel by a second operation, performed when the obstruction has been overcome and the patient in good condition. It should be remembered that the hyperplastic form of the disease is sometimes met with as part of a more widespread involvement of the intestine. Search of the remainder of the bowel is therefore advisable in every case, before the nature and extent of the operation is decided.

AMŒBIASIS

THE surgeon who works in an area where amœbic dysentery is endemic will soon become familiar with the manifestations of the disease. However, sporadic cases of amœbic infection may be seen in temperate zones, particularly when so many men and women served during the recent war in tropical areas, and it is therefore important that surgeons should be familiar with certain aspects of the disease. A definite history of dysenteric infection is not always obtained, and rarely an affected patient will be seen who has never been abroad.

In acute cases, free perforation of an ulcer in the colon or a walled-off perforation forming a peri-colic abscess may sometimes require surgery. The diagnosis in these cases is seldom in doubt. More confusing is the patient with an amœbic infection of the cæcal region, who may present with great clinical resemblance to appendicitis, often without diarrhœa. Appendicectomy is dangerous, and in endemic areas the stools should be examined for amœbæ before operating for appendicitis, and anti-amœbic drugs given if there is any past history of amœbiasis. If the correct diagnosis is not made until the abdomen has been opened, it should be closed again without removing the appendix.

Chronic amœbic infection of the colon seldom results in a diffuse colitis, but tends to involve particularly the cæcal and recto-sigmoid regions. Sometimes a granuloma (amœboma) will form and lead to a palpable mass, but frequently the infection leads only to a fairly localized granulomatous thickening of bowel wall, which can only be detected by rigidity or mucosal changes on X-ray examination. In either case diagnosis may be difficult, as bowel symptoms are seldom marked, and the amœbæ may be found in the stools only after prolonged and repeated search. A normal rectal mucosa on sigmoid- [illegible]

[illegible] clears up rapidly on anti-amœbic drugs, a therapeutic test may be of value if there is doubt. A very small percentage of cases of amœbic dysentery may be followed, perhaps after several years of normal health, by chronic ulcerative colitis. This may also occur following bacillary dysentery, but it does not follow that ulcerative colitis, even in these cases, is due to chronic dysenteric infection.

DIVERTICULOSIS AND DIVERTICULITIS

DIVERTICULA of the intestinal tract may be congenital or acquired. The former are comparatively rare, and are hardly ever seen in the colon; the most common is the Meckel's diverticulum of the ileum. Acquired diverticula, on the other hand, are more common in the colon than in any other part of the intestine, and not infrequently lead to complications.

Diverticulosis of the colon was probably first described by Cruveilhier in 1849, but it is only since the advent of X-rays that the condition has been fully studied and its fre-

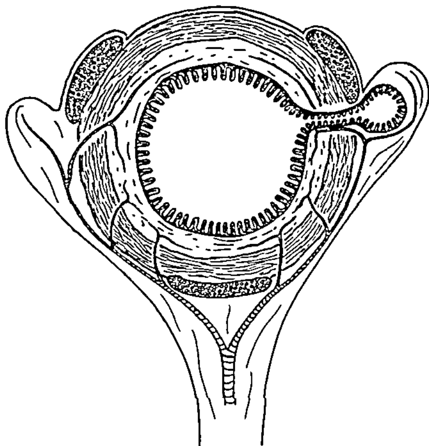


FIG 179 Diagram to show the normal anatomy of the vessels in the sigmoid colon and the formation of diverticula The latter come into close relationship with the tæniæ coli

quency and importance realized. The disease is mainly one of advancing years, being rare under 40 and most frequent in those over 60. Men and women appear to be equally affected by diverticulosis, but the inflammatory changes of diverticulitis and the complications that arise therefrom are commoner in men.

Incidence and Ætiology

The true incidence of this condition is difficult to assess as it depends on radiological studies. It has been estimated that about 5 per cent of people over 40 years of age have diverticulosis, and of these it is likely that between 10 and 15 per cent will develop some degree of diverticulitis.

Probably two factors are concerned in the formation of diverticula, namely weak spots in the intestinal wall, and periods of raised intra-luminal pressure, the two together resulting in a herniation of the mucosa through the muscle coats. The weak spots are in nearly all cases the defects in the muscle coats where the blood-vessels pierce the wall to gain the submucous plane. These vessels pierce the wall in a very constant position, just on the mesenteric side of the two lateral tæniæ coli, and it is an observed fact that diverticula occur in two parallel rows in this situation. The appendices epiploicæ are also situated in that quadrant of the bowel, so that the diverticula often enter the appendices (Fig. 179). The raised intra-luminal pressure is probably brought about by irregular spasm of the muscle coat (instead of the normal co-ordinated peristaltic wave) resulting in momentary elevations of pressure in localized segments of bowel. Obesity and constipation may possibly be aggravating factors in some cases, but are of little importance as primary causes (Edwards, 1939).

Pathology

In the wall of a congenital diverticulum, all coats of the bowel are present. Acquired diverticula, on the other hand, are herniations of the mucous membrane through the muscle coat, and in their walls are to be found only the mucosa, submucosa, and some muscularis mucosæ, and a serosal covering. Though diverticula may occur throughout the colon, they are much more common on the left side, particularly in the sigmoid, though diverticula of the rectum are very rare. Similarly, diverticulitis is usually confined to the descending and sigmoid colons, probably because of the solid nature of the bowel contents in this part.

The diverticula are usually flask-shaped, with a narrow neck through the muscle coat and an expanded body. It is this shape and the absence of a muscle coat which probably accounts for the complication of inflammation leading to diverticulitis. Fæces enter the sacs, become inspissated forming hard pellets, and cannot then be extruded. The mucosa of the sacs becomes traumatized, and organisms may thus enter the tissues. Acute inflammation may follow, which will either resolve or progress to abscess formation depending on the resistance of the patient and the treatment given. The more common result, however, is the gradual development of a chronic peridiverticulitis, which will in time lead to the formation of much fibro-fatty tissue round the affected bowel, causing thickening of the wall and narrowing of the lumen. This tissue may extend over several inches of the colon resulting in a firm sausage-shaped mass, and may become adherent to other viscera or to the posterior abdominal wall.

Clinical Features

Diverticulosis is a symptomless condition, which is only revealed by barium enema examination. The spasm of the colon frequently present in such cases may sometimes give rise to abdominal discomfort and constipation.

In the acute stage, diverticulitis may present with pyrexia and pain in the left side of the abdomen, and examination will reveal tenderness over the colon, with rigidity and a palpable tender swelling in the more severe cases. Rarely, a patient may first present with peritonitis following perforation of an inflamed diverticulum.

The usual symptom of the more chronic peridiverticulitis is again pain in the left iliac fossa, often brought on by exertion or jolting movements. Some alteration of bowel

habit is common, constipation being the more usual though there may be periods of diarrhœa. Abdominal colic, a sense of distension, and dyspepsia are frequent symptoms. Bleeding is unusual, but is said to occur in about 15 per cent of cases; it would always suggest the possibility of a co-existing neoplasm. Physical examination of such a patient seldom reveals anything abnormal apart from a little tenderness in the left iliac fossa, but a thickened coil of bowel may sometimes be felt on abdominal or rectal examination.

Diagnosis

The recognition of diverticulosis and diverticulitis rests essentially on radiological examination after an opaque enema. The earliest change noted is a narrowing and rigidity of the bowel outline with a finely serrated "ripple border"—the so-called pre-diverticular state. This is followed in time by fully developed diverticulosis. The latter is characterized by the typical sacs arising from the bowel, often best seen in the post-evacuation film; the barium shadow may have a crescentic outline if there is a concretion in the sac (Fig. 180). In cases of diverticulitis where there has been pericolic fibrosis, X-rays will usually show an irregular filling defect, often with well-formed diverticula in nearby bowel (Fig. 181). The distinction between such a defect and that caused by a carcinoma may be difficult; in the latter the defect is characteristically shorter and with more abrupt margins, and the mucosal destruction may be discernible, but certain distinction is not always possible.

Sigmoidoscopy in this disease is mainly of negative value in excluding carcinoma and colitis. Rigidity of the lower sigmoid, with narrowing of the lumen and angulation of the bowel may be noted and suggest the correct diagnosis, and exceptionally the opening of a diverticulum may be seen. Visible blood on the wall of the bowel, especially if bleeding has been one of the patient's symptoms, would strongly suggest a co-existing neoplasm, and is usually an indication for surgical treatment.

Differential Diagnosis

Diverticulitis by its varied manifestations may simulate many other diseases. Acute diverticulitis may resemble other acute intra-abdominal inflammations, particularly *appendicitis* and *pelvic inflammatory disease.* The more common form of peridiverticulitis with a localized narrowing of the bowel may closely simulate *carcinoma*, and the X-ray and sigmoidoscopic distinctions between the two conditions have been mentioned. It must not be forgotten that diverticulitis and carcinoma may both occur in the colon, though diverticulitis should not be regarded as a pre-malignant condition. *Hyperplastic tuberculosis* and *regional colitis* are both rare, but should be kept in mind as possibilities when X-ray findings are atypical.

The rare occurrence of inflammation in a cæcal diverticulum is seldom correctly diagnosed. Such cases closely resemble acute appendicitis clinically, and come to operation with that diagnosis: the mass disclosed is usually then regarded as malignant and a right hemicolectomy performed.

Complications

Free Perforation. This is comparatively rare, as the pericolic fat and omentum usually contrive to seal off an inflamed diverticulum before it can perforate. If it does occur, the signs of a perforated viscus and peritonitis result.

Abscess. An inflamed diverticulum that becomes sealed off from the bowel may progress to form an abscess. Pain with pyrexia and toxæmia will be the main clinical manifestations, and though a mass will form in relation to the colon it may not be palp-



(*By courtesy of Dr. Norman Henderson*)

FIG. 180 Barium enema in a case of diverticulosis. In square 1 the "ripple border" of the pre-diverticular state is well seen, while in square 2 the diverticula are fully formed. In square 3 an intermediate stage is seen, in which the necks are still narrow Note that different stages may occur in different parts of the bowel at the same time

able due to obesity or overlying muscular rigidity. Such an abscess may approach the surface and rupture or be drained externally, or may rupture into the peritoneal cavity or a neighbouring viscus. The latter course will lead to the formation of an internal fistula, while an external fistula will usually result from surface drainage. Occasionally

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considerable narrowing of the colon is produced in many cases and the resulting partial obstruction is one of the most frequent indications for surgery.

Fistula. This may arise from an abscess as mentioned above, or more insidiously from a low-grade inflammatory process. An *entero-colic fistula* between small bowel and colon will cause diarrhœa and will soon lead to nutritional defects and weight loss. More common and more serious is a *vesico-colic fistula.* Diverticulitis of the colon is the most common cause of this fistula, though it may also arise from cancer of the colon, more rarely cancer of the bladder, and from pelvic abscesses following appendicitis. It is uncommon in women because of the presence of the uterus.

Clinically, there may be a history of previous attacks of acute diverticulitis, with recent frequency and dysuria. When the fistula first forms, there is usually an intense cystitis with severe symptoms, but an extraordinary degree of immunity is soon developed by the bladder, and the cystitis largely resolves. Pneumaturia, usually intermittent, is the classical symptom of an established fistula, and fæcal particles may be recognized in the urine. It is rare for urine to find its way into the bowel and be passed per anum. In spite of the infection in the bladder, ascending infection of the urinary tract is unusual. The diagnosis of the fistula depends mainly on the history, cystoscopic examination, and the barium enema; cystograms may also be of value but it is rarely possible to demonstrate the actual fistula radiologically. It is important for the surgeon not only to diagnose the fistula but to ascertain its cause, and particularly to recognize malignant disease if it should be responsible. It must not be forgotten that intestino-vesical fistulæ may also come from the small bowel, for instance in cases of Crohn's disease.

In women, fistulæ into the vagina or into the Fallopian tube may rarely occur.

Treatment

Medical Treatment

The aim of treatment in cases of diverticulosis and mild cases of diverticulitis is the prevention of progression of the disease and the avoidance of complications Towards the attainment of these ends it seems logical to advise a bland low-residue diet, devoid of pips and seeds; and to prescribe liquid paraffin in sufficient dose to ensure a soft motion. In cases with spasm of the colon the administration of anti-spasmodics and sedatives in small doses may be helpful. On this regime the large majority of patients will continue to have little or no trouble from their disease. The dangers of perforation of a diverticulum following violent purgation or as a result of enemata or colonic lavage should be impressed on the patient

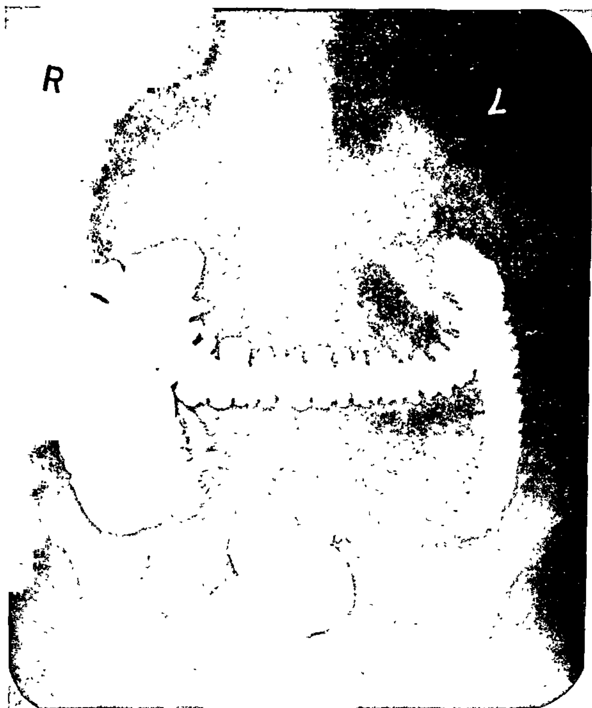
In cases of *acute diverticulitis* the treatment is again medical in the first instance. Rest in bed is essential, and paraffin should be given as above. Treatment with sulphonamides or antibiotics should be started immediately, and in most cases satisfactory resolution will be observed. Some cases will progress to abscess formation in spite of treatment and require surgery, which should in the first instance be limited to drainage only. A fæcal fistula sometimes follows, and a proximal colostomy or resection of the diseased segment may be necessary later.

Surgical Treatment

Surgery in this disease is required mainly for its complications, but the increasing safety in recent years of colon surgery has naturally tended to widen the indications (Lloyd-Davies, 1953).

such an abscess may track extensively before coming to the surface and thus may present in the most unusual situations, such as buttocks, thighs, or loins.

Intestinal Obstruction. Small gut obstruction may result from adhesions of a loop to an inflammatory mass, or the colon may become obstructed due to progressive narrowing from peridiverticular fibrosis. Neither commonly causes a complete obstruction, but a



(Mr W. B Gabriel's case)

Fig 181. Barium enema showing a "filling-defect" in the sigmoid colon due to diverticulitis Note the length of the filling-defect and its serrated outline, and the diverticula in the bowel above.

Vesico-colic Fistula

The surgical correction of this complication may be a difficult undertaking, and it is always advisable to establish a transverse colostomy as a first step. The distal bowel can then be cleared by suitable washouts and the infection controlled. It is probably wise to allow an interval of about 6 weeks before attempting resection; a fistula will often close spontaneously in this time, though it would almost certainly recur if the colostomy were to be closed without removal of the diseased segment of bowel. At the main operation, the bladder and bowel are separated, the diseased bowel is resected and continuity restored. The opening in the bladder is usually on the posterior wall, and must be carefully closed, the bladder being then drained by indwelling catheter for several days. After a few weeks interval the colostomy can be closed.

Colostomy. In cases of severe diverticulitis where it is considered that the patient is not fit enough to withstand a major operation, a permanent transverse colostomy will often be required. A good spur ensuring complete diversion of the fæcal stream is essential, or it is often better to separate the two ends of bowel by a strip of skin (Devine, 1938). Even with such a colostomy, complete quiescence of the inflammation is not always obtained, and cases have been reported in which a vesico-colic fistula has formed in spite of a proximal colostomy. In most cases, however, the patients remain well and free from symptoms, and the disease gives no further trouble.

It sometimes happens that a patient may present with a mass in the pelvis, in which even after full investigation certain distinction between diverticulitis and carcinoma cannot be made, or in which the presence of a carcinoma in an area of diverticulitis cannot be excluded with certainty. Laparotomy reveals a pelvic cavity full of firm tissue binding all viscera together, but often the diagnosis remains obscure. The treatment of such a case requires much judgment. Where excision is possible without damaging other viscera or endangering life, it is best carried out; but where the extent of the mass prevents this, a proximal diversionary colostomy is usually the best immediate procedure. The diagnosis may soon become apparent after such diversion, and if malignant disease is found to be present, a later determined attempt at its removal may still be made.

ULCERATIVE COLITIS

Ulcerative colitis is a disease characterized by diffuse inflammation of the large bowel, with ulceration of varying degree, for which as yet no single ætiological factor is known. Other forms of colitis, such as the dysenteries due to specific micro-organisms, are excluded by the definition, leaving a group of cases of non-specific colonic inflammation to which the term ulcerative colitis is applied. The disease is variable in its extent, severity, and clinical course, and our knowledge of many aspects of it is still far from complete.

Men and women appear to be affected equally. In over 80 per cent of cases the onset of the disease has been before the age of 40; most people affected are in the twenties and thirties, but the disease does occur in children and in old age.

Ætiological Factors

Various theories as to the cause of the disease have been put forward, none of them entirely satisfying. The similarity to the infective diarrhœas has naturally led to the

Chronic Diverticulitis with Pericolic Fibrosis

This is now the most common indication for surgery. Patients with severe or increasing symptoms not responding to medical management, patients with recurrent attacks of acute diverticulitis, and patients with progressive narrowing of the bowel are usually best treated surgically. In the same way, patients with increasing urinary symptoms not due to vesical or prostatic disease are probably in danger of developing a vesico-colic fistula and should be advised surgery.

Until recent years, a proximal colostomy was all that could be done with any safety for most patients. This often needed to be permanent, for closure, even a year or more after complete freedom from symptoms, usually led to a return of activity in the diseased colon, often requiring re-establishment of the colostomy. However, it is now usually possible to resect the diseased segment and restore continuity with safety, and this is now the surgical aim. Technically, it may be a difficult procedure, due both to the widespread inflammatory fibrosis often present, and to the extensive length of colon which may be diseased. The choice of procedure in every case must depend on the particular conditions; a three-stage operation—transverse colostomy, resection and anastomosis, closure of colostomy—is necessary in obstructed cases and those with acute inflammation or much œdema, but a one-stage resection with primary anastomosis is suitable in most cases, and should certainly be done when there is any suspicion of malignancy. The Paul-Mickulicz operation is seldom possible because of the length of colon involved and the rigidity and shortening of the mesentery.

If a staged procedure is adopted, the colostomy should be established in the right half of the transverse colon, and should be so constructed as to ensure complete diversion of the fæcal stream. The interval between the establishment of a colostomy and the resection must again vary with each case; after 3–4 months little further resolution of inflammation is to be expected, and fibrosis may increase and add to the difficulties. It is seldom necessary to wait as long, 3–6 weeks being a satisfactory interval in most cases. The resection must include the whole length of the inflammatory process, and if possible all bowel showing diverticula, though the extent of diverticulosis may prevent the latter. It will often be necessary to remove the whole left colon and anastomose the transverse colon to the upper rectum, hence the necessity for siting any transverse colostomy well to the right. The latter can usually be closed two weeks after the resection.

Perforation

Surgery is necessary as an emergency. It is best to make no attempt to mobilize the colon with a view to exteriorization, as the necessary dissection is likely to be time-consuming and dangerous in a sick patient. The hole should be closed by sewing fat, and omentum if available, over it, and the site drained with soft rubber; the peritoneal cavity should then be sucked dry, and a right transverse defunctioning colostomy established. Resection will probably be necessary later.

Obstruction

A colostomy in the right transverse colon should be established as a preliminary to a later resection. The distinction between diverticulitis and carcinoma may be impossible at the emergency operation, and the second stage should not be delayed if there is any doubt.

disease is proceeding (Sammons, 1951). Another enzyme, lysozyme, normally found in saliva, tears, and bile, is found in the stools of many patients with colitis, but there is little evidence that it is of ætiological significance, and it is probably secondary to the purulent exudate.

Pathology

Extent of Disease. In nearly all cases of ulcerative colitis the rectum and recto-sigmoid are affected, and are usually the parts most severely diseased. The disease appears in



(Mr H R Thomson's case)

FIG. 183 Specimen removed by sub-total colectomy from a patient with ulcerative colitis. Note the shortening of the bowel, the loss of sacculation and the thick wall. The mucosa is dark and granular, with patchy ulceration on the left side

most cases to start there and extend proximally; the rate at which it does so varies greatly in different patients, and thus patients may be seen with only the rectum involved, or with the left colon and rectum involved, or with the whole colon affected. Rarely the lowest part of the ileum is also diseased, and in such cases the ileo-cæcal valve is rigid and patent.

A small number of cases are seen in which only a segment of the colon is involved without disease of the rectum and lower sigmoid. The affected part may be anywhere in the colon and may sometimes be associated with similar changes in the lower ileum, either in continuity or with intervening normal bowel. It has been suggested that these

suggestion that the disease is due to infection by a specific organism or virus; but none of the claims made in the past for the existence of such an organism have been substantiated by further research. An allergic response to certain articles of diet has been suggested, but in most cases there is no evidence that allergy plays any significant role. Considerable attention has been paid to the personality and emotions of the individual, and in some patients with colitis a definite psychological pattern is found, of which the outstanding features seem to be above-average intelligence, extreme sensitivity, lack of aggressiveness, and emotional immaturity. It appears likely that colitis is a "stress" disease, and that emotional conflict may be a factor both in the original onset of the disease and in relapses.

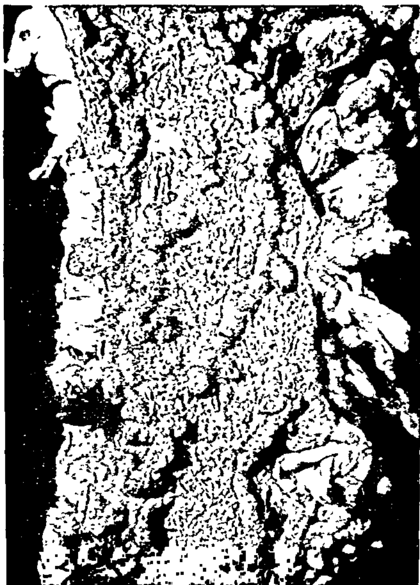


FIG. 182. Photograph of part of the colon in a case of ulcerative colitis There is extensive ulceration with surviving scattered tags of œdematous mucosa

Recently, attention has been paid to certain enzymes (mucinases) which have a solvent effect on the mucus of the colon, and further research as to their nature and role in this

nearly always be done with little disturbance to the patient. The sigmoidoscopic picture in colitis varies with the state of the disease. In the mild cases, the mucosa shows a pink finely granular appearance with loss of the normal vascular pattern; in more active cases the mucosa is darker red in colour, bleeds easily if touched, and its granularity is more obvious. In the most severe cases, the above changes are still more marked, and thickening and rigidity of the bowel with narrowing of the lumen from œdema are seen. Ulceration may be seen as well as the granularity, but in the majority of cases of so-called ulcerative colitis no discrete ulcers can be detected with the sigmoidoscope. A variable amount of blood, mucus, and pus is seen in the lumen and covering the wall. In cases of longer duration, the presence of pseudo-polyps, strictures, or neoplastic change must be looked for. The instrument should if possible be passed to full length, and the changes noted at different levels; it is not uncommon in mild or early cases to find that the changes are confined to the rectum, with normal mucosa above 15 or 20 centimetres.

Barium enema X-ray should be done in every case. Here again the picture varies greatly, both with the state of the disease and the area involved. The characteristic changes to be looked for include loss of mucosal pattern, giving a roughened outline to the shadow; loss of haustration, causing a "lead-pipe" outline to the colon; and contraction of the colon, resulting in lowering of the flexures and shortening of the whole bowel (Fig. 184). Should there be involvement of the terminal ileum it will usually be demonstrated by the barium enema, but if there is any clinical suspicion of small bowel involvement a barium meal and follow-through should also be requested.

Laboratory procedures are seldom helpful in diagnosis, but are necessary for full investigation. The blood-count shows anæmia of varying degree, and there is often some polymorphonuclear leucocytosis. The sedimentation rate may be raised. In severe cases there may be marked changes in the blood chemistry due to the loss of fluid, electrolytes, and protein from diarrhœa. The finding of steatorrhea suggests extensive disease of the small bowel. It is of course essential to exclude the dysenteries by careful and repeated examination and culture of the stools.

Differential Diagnosis

The diagnosis of ulcerative colitis rests on three things: the clinical history, the sigmoidoscopic appearance, and the barium enema, of which the first two are the most essential. If this is appreciated, few diseases are likely to cause confusion. The infective diarrhœas must be excluded as mentioned above, particularly in fulminating cases. Cases with pseudo-polypi must be differentiated from multiple polyposis, in which disease the mucosa between the polyps is normal. Neoplasm of the colon, Crohn's disease, and hyperplastic tuberculosis may cause difficulty in "regional" cases.

Complications

Systemic Complications. Various *skin affections* are seen, of which the most usual is Erythema Nodosum. Skin lesions from vitamin deficiencies may occur in severe cases, and rarely a spreading skin gangrene has been reported. Ulceration of the legs is sometimes seen. *Arthritis*, usually of "rheumatoid" type, is seen in 5–10 per cent of cases, not infrequently associated with erythema nodosum, and affecting the smaller joints. *Nutritional deficiencies* are usual in chronic cases, and anæmia, hypoproteinæmia, and vitamin

cases of "regional colitis" are different in origin from cases of true ulcerative colitis, and are more akin to Crohn's disease (Wells, 1952). The present imperfect state of our knowledge regarding both these diseases and their relationship to each other allows only of speculation on this point.

Changes in the Bowel. The early stages of the disease are marked by congestion and œdema of the mucosa, giving the bowel lining a red and granular appearance. Patches of ulceration may then appear and coalesce, so that irregular islands of congested mucosa may be left between shallow ulcers having a floor of granulations. The other coats of the bowel show increased vascularity and œdema, but at this stage the main changes are confined to the mucosa and the superficial layers of submucosa. If the process continues, the ulcers enlarge at the expense of the surviving mucosa, and only irregular tags of mucous membrane, infiltrated with inflammatory cells, may be left scattered through the colon (Fig. 183). These tags enlarge from œdema and hyperplasia, and form "pseudo-polyps," showing considerable variation in size and shape. The muscle coats become hypertrophied and the bowel lumen contracted, and in cases of long standing fibrosis of moderate degree occurs (Fig. 184). If at any time the disease becomes quiescent, a considerable amount of repair may take place; a thin layer of epithelium grows from the remaining islands of mucosa over the denuded areas, though the normal arrangement of mucosal crypts is probably never regained.

Clinical Features

The onset of the disease is usually insidious, sometimes acute. Diarrhœa is usually the first and certainly the most common symptom; mucus and blood are usually noticed in the motions before long, and in severe cases the stools may consist of little else. Lower abdominal colic is frequent. Constitutional disturbance occurs in severe cases with pyrexia, malaise, loss of weight and strength, anæmia, and often hypoproteinæmia.

It is useful to have a clinical classification, and though this disease is notoriously variable in its course and severity in different individuals, most cases fall into one of the following groups:

(1) *Acute Fulminating Type.* This is an uncommon but dangerous form of the disease. It may progress to death in a few weeks, or become chronic and continuous, or improve to complete remission.

(2) *Chronic Continuous Type.* The onset may be insidious, or this may follow an acute onset. The disease may continue for years, with variation in intensity, but never with complete relief of symptoms The patients often become semi-invalids, and an acute fulminating episode may occur at any time.

(3) *Chronic Relapsing Type.* In these patients, periods with symptoms of varying severity are separated by periods of complete freedom, the latter varying in duration from weeks to years.

Diagnosis

Examination of the patient must include an assessment of the state of nutrition, the degree of constitutional disturbance, and the presence of skin complications. On abdominal examination, the contracted colon may sometimes be felt, or tenderness in the line of the colon be elicited.

Rectal examination is most important, and must include sigmoidoscopy, which can

deficiencies often go together. Various degrees of *liver damage* occur in long-standing cases, probably due to the impairment of nutrition.

Colonic Complications. The formation of *pseudo-polypi* is common, but should be regarded as the end-result of a severe ulcerative process rather than as a complication. *Perforation* of the bowel is rare; it occurs in patients already acutely ill, and is often fatal. *Severe hæmorrhage* occurs sometimes, usually in patients with a marked degree of pseudopolyposis. *Stricture* may result occasionally, particularly in the rectum; various degrees of narrowing of the colon may be seen in X-rays, but these are more usually due to muscular spasm and hypertrophy than to fibrosis. *Abscesses and fistulæ* in the anal region may occur at any stage of the disease. They are often indolent, and cure is difficult while the disease persists. Abdominal fistulæ are rare, but recto-vaginal fistulæ in women are not infrequent.

There is now an impressive body of evidence to show that *carcinoma* occurs with considerably increased frequency in the bowel of patients with ulcerative colitis, particularly in those who have had the disease for several years (Counsell and Dukes, 1952). The exact incidence of malignant change is difficult to compute, but probably 25 per cent or more of patients with severe disease of over 10 years duration will develop carcinoma of the large bowel. Unfortunately there may be little clinical or radiological evidence of the malignant change, as the carcinoma is usually intra-mural with little surface protuberance or ulceration, and any symptoms are liable to be attributed to an exacerbation of colitis. Further, these tumours complicating colitis are usually anaplastic colloid carcinomas of high malignancy, and metastasis to lymph-nodes, omentum or liver has often already occurred by the time the diagnosis is made. Not infrequently multiple foci of carcinoma may develop at the same time in the affected colon.

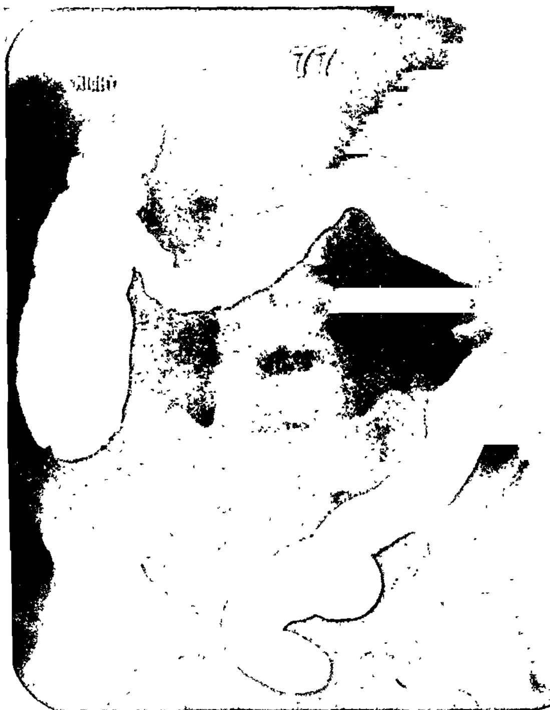
Treatment

Medical Treatment. In the absence of any specific therapy for this disease, medical treatment aims at the maintenance of the patient's general health and resistance, in the hope of inducing a remission of symptoms. The measures employed include rest; adequate sedation to ensure sleep, which may need to include drugs to diminish diarrhœa; high-calorie high-protein diet with minimal residue, and the provision of adequate fluid, minerals, and vitamins; and the maintenance of hæmoglobin above 70 per cent by iron therapy and repeated small transfusions if necessary. *Antibiotics* and insoluble *sulphonamides* are rarely of great benefit, but they may help to overcome secondary infection and toxæmia in severely ill patients. *A.C.T.H. and Cortisone* have been used recently in various stages of the disease, and appear to be of value in that the chance of remission is increased with their use. A recent controlled study (Truelove and Witts, 1954) shows that Cortisone is more likely to benefit a patient in the initial attack than in a relapse, and is of most value in the more acute cases. Not all cases respond, and in those that do, relapse may occur when treatment is stopped Moreover, hæmorrhage and perforation have been reported as occasional complications during treatment. Further experience with these drugs is necessary before their value in this disease is really known.

Indications for Surgery

(1) Chronic Invalidism

This is now the most frequent indication for advising surgery. It includes patients with chronic continuous disease who are losing ground, and those with relapsing disease

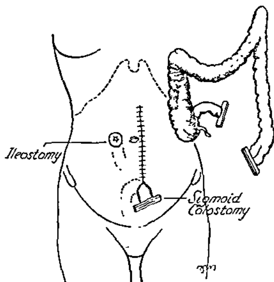


(Mr W B Gabriel's case

Fig. 184 Barium enema in a case of ulcerative colitis, showing the "lead-pipe" outline and the rough mucosa Note the lowering of the splenic flexure

ILEOSTOMY AND PROCTO-COLECTOMY

The operation is usually best done in two stages: in the first, the terminal ileostomy is established, and the colon removed from the cæcum to the lower sigmoid, with exteriorization of the distal end of the latter (Fig. 190); in the second, the remaining recto-colic segment is removed by abdominal and perineal dissection. This technique is in most cases preferable to that formerly practised whereby the ileostomy was done as a separate procedure before the two-stage colectomy. Particularly is this so in the desperately ill patients where operation is undertaken to save life: ileostomy alone in such patients carried a formidable mortality, probably because the disturbances of fluid and electrolyte balance common with an ileostomy were added to the toxic absorption and fluid loss from the colon (Ripstein *et al.*, 1952). These patients stand a colectomy to the mid-sigmoid well (it is often a relatively simple operation, as the contraction of the bowel renders the flexures more accessible) and being relieved of their toxæmia, there is usually rapid improvement in general health.



(Counsell, P B & Goligher, J C, Lancet 1952, 2, 1045)

FIG 185 Ileostomy and subtotal colectomy in one stage, showing the length of bowel removed, and the establishment of the two stomas The clamp is removed from the sigmoid colostomy after a few days

The excision of the remaining recto-colic segment is best delayed for a few months until there has been a great improvement in general condition It may be done sooner if persisting arthritis, continued hæmorrhage, or other reason exists. Alternatively, in selected patients in good general condition the entire procedure (ileostomy and procto-colectomy) may be done in one stage, an operation which sounds formidable, but is well tolerated if expeditiously performed.

ILEOSTOMY

This is a necessary accompaniment of most operations performed for removal of the diseased bowel, but is rarely done now as the sole surgical procedure. While it does completely divert all fæcal contents from the colon, and often produces a wonderful clinical improvement, the diseased bowel, with all its potentialities for trouble, is left. (The management and complications of ileostomy are discussed on p. 452.)

COLECTOMY WITH PRESERVATION OF THE RECTUM

This operation aims at removing most of the diseased bowel, while retaining rectal control; the colon is removed and the ileum joined to the rectal segment either by an enterotome method or by a sutured anastomosis, and the rest of the large bowel excised (Devine and Devine, 1948, Corbett, 1952).

While good results have been claimed for these procedures it must be remembered that the rectum is usually the most severely diseased part of the bowel, and complications such as fistulæ, stricture, and even malignant change are still likely to occur, and have

whose intervals of freedom are becoming shorter. The length of history is of little value in deciding the need for surgery, because the rate of progress varies greatly in different people. It seems certain, however, that the longer the duration of the disease the greater is the risk of carcinoma developing; hence a long history in a case of any severity would be an added reason for surgical treatment.

It is becoming recognized that surgery in this disease has much to offer and should not be delayed overlong. However, surgery will in most cases mean that the patient will have to accept the disability of a permanent ileostomy. Thus a considerable mental adjustment is necessary, which may be more difficult for the patient to make unless all medical measures have been tried without success, and unless the disease has been severe and disabling and of some duration, so that the improvement in health after operation is likely to be striking. Greater co-operation between surgeon and physician is necessary and patients who are obviously losing ground should be advised surgery while still in good condition.

(2) Severe Acute Episodes

Patients with acute fulminating disease and those with an acute exacerbation of relapsing disease may go rapidly downhill in spite of all medical measures, and surgery may be necessary to save life. Such cases require the most careful judgment in deciding the right time to operate.

(3) The Presence of Complications

Arthritis occurring with ulcerative colitis will not clear up until the diseased bowel has been removed, and is an indication for early ileostomy and colectomy.

Of the complications affecting the bowel, *perforation* naturally calls for immediate operation; ileostomy and colectomy should be done if possible. *Hæmorrhage* may rarely call for emergency colectomy. the presence of *anal abscesses or fistulæ* and of *strictures* are seldom indications by themselves, but would be an added reason for surgery in a patient with chronic disease. The presence of *carcinoma* is an obvious indication, but the chances of long survival are slender. The colon should be removed before carcinoma develops.

(4) "Regional" Cases

Most such cases, particularly those confined to the colon, do well after resection with anastomosis. The ultimate prognosis should be guarded, however, as recurrence in another part of the bowel, or later diffuse involvement of the colon, are not unknown.

Choice of Surgical Procedure

In the present limited state of our knowledge about this disease, it is unfortunate that surgery to be effective has also to be disabling, in that ileostomy is nearly always necessary and once established must usually be permanent. It is now realized that ileostomy alone, though diverting the fæcal stream, does not always lead to quiescence of the disease and healing of the colon. Toxæmia, hæmorrhage, protein-loss, and arthritis may all continue, and the patient is never free from these risks until the colon has been removed; nor must the considerable incidence of malignant change in these diseased colons be forgotten. For these reasons, the establishment of a terminal ileostomy with excision of the entire large bowel is now considered to be the correct treatment for most cases of ulcerative colitis requiring surgery (Cattell *et al*, 1953)

5 per cent. Few reliable figures about the natural history of the disease and its mortality with medical management have been published, but it is probable that a considerably higher mortality results; 20 per cent of patients are said to die within a year of the onset of the disease (Rice-Oxley and Truelove, 1950). Moreover, after surgery, patients are restored to robust health and are able to resume their former work and social activities. Such a life, even with an ileostomy, is far preferable to that of chronic invalidism.

BENIGN TUMOURS

Connective Tissue Tumours

CONNECTIVE tissue tumours of the colon are rare. *Lipomas* and *Leiomyomas* occur, and usually cause symptoms of partial bowel obstruction. The tumour may become pedunculated and be the cause of an intussusception; congestion may cause rectal bleeding, and thus the clinical resemblance to carcinoma can be striking, especially as a filling defect is seen with opaque enema. Resection of the involved part of bowel results in cure.

Hæmangioma may be circumscribed or may involve a large part of the bowel and mesentery. It usually causes rectal bleeding, sometimes severe, in a young patient. The diagnosis may be made by sigmoidoscopy or barium enema, or may remain obscure until disclosed by laparotomy. Treatment is by resection if possible, but if the lesion is too extensive, ligature of the main feeding vessels may result in cessation of further bleeding. Diversion of the fæcal stream by colostomy or ileostomy will help to prevent bleeding if the tumour is too widespread for removal.

Endometriomas may involve the colon from the serous surface, a plaque of connective tissue stroma containing uterine glands gradually narrowing the lumen without invading the mucosa. Symptoms of obstruction result and sometimes rectal bleeding; a filling defect is often apparent on X-ray and thus resemblance to carcinoma is strong. A limited resection is necessary for cure.

Epithelial Tumours

Epithelial tumours of the colon arise much more frequently than the above, and are of particular importance because of their tendency to become malignant. Isolated tumours may occur, or there may be a small number, of varying size, scattered throughout the large bowel. (The term Polyposis should be reserved for the familial disease in which large numbers of polypi develop at an early age, and which is described in the next section.)

These tumours may arise in any part of the colon, but are most common in the sigmoid, and most patients are over 40 years of age. Both adenomas and papillomas occur: the former are the more common and are usually compact tumours which grow from a small area of mucosa and therefore tend to become pedunculated, while the rarer papillomas have their origin from a wider area of mucosa, and are less likely to become polypoid. Both appear to have an equal tendency to malignant change, and it is important to realize that the carcinoma that develops is not necessarily of low malignancy, but may metastasize early. Such tumours may act as the starting-point of an intussusception,

indeed done so in some reported cases (Gabriel, 1952). Nevertheless, there has recently been a revival of interest in operations preserving the rectum, and satisfactory results are claimed (Aylett, 1954). Further experience and a longer follow-up will be needed before the procedure can be properly assessed.

Colectomy with removal of the rectum and ileoanal anastomosis, while satisfactory on pathological grounds, seldom allows good control, and an abdominal ileostomy is usually preferable.

Colostomy

It not uncommonly happens that the need for surgery arises when only the left half or less of the colon appears to be affected by the disease. In such cases the surgeon may be tempted to establish a transverse colostomy instead of an ileostomy (with excision of the affected part of colon if necessary). However, it is often extremely difficult to determine radiologically or at laparotomy the precise proximal extent of the disease; it must be remembered moreover that even if it is confined to the left colon, it may later spread to the right colon, and this progress does not appear to be delayed by the establishment of a colostomy. A subsequent ileostomy has therefore frequently to be performed in these cases, and for this reason transverse colostomy is usually better avoided.

Appendicostomy and Cæcostomy

These were in former years common procedures, by means of which irrigation of the bowel could be carried out. It is doubtful if such treatment alters the natural course of the disease, and they are now rarely employed.

Vagotomy

Since this operation was introduced for the treatment of peptic ulcer, it has also been advocated for the treatment of ulcerative colitis. Reports have been favourable in many cases, but it is agreed that good results can only be expected in early cases, before irreversible changes have taken place in the colon. However, the natural course of this disease is so often one of remission and relapses that some years must elapse before the results of vagotomy can be truly assessed.

Resection and Anastomosis

This is the treatment of choice for most cases of regional colitis. The abdomen must be carefully explored before embarking on the resection, as "skip-lesions" in the small intestine are sometimes seen, and their presence may alter the line of treatment and the prognosis.

Results

In former years, physicians were reluctant to advise patients with ulcerative colitis to undergo surgery, both because of the high immediate mortality, and because an ileostomy was such a serious disability. Recent years, however, have seen a considerable reduction in the hazards of operation and great improvement in the design of ileostomy appliances, making these objections less serious. With increased experience has come the realization that there is an optimum time for surgery, and that to delay operation until the patient is desperately ill is certain to result in increased mortality. In experienced hands, and with proper selection of cases, mortality for the whole procedure should not exceed

5 per cent. Few reliable figures about the natural history of the disease and its mortality with medical management have been published, but it is probable that a considerably higher mortality results; 20 per cent of patients are said to die within a year of the onset of the disease (Rice-Oxley and Truelove, 1950). Moreover, after surgery, patients are restored to robust health and are able to resume their former work and social activities. Such a life, even with an ileostomy, is far preferable to that of chronic invalidism.

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and indeed it is rare to see an intussusception in an adult without a tumour at the apex.

Bleeding is usually the only symptom caused by a polyp in the colon. Villous papillomas often cause diarrhœa with excess of mucus. Small polyps may not give rise to any symptoms, and those in the lower sigmoid colon are frequently detected by a routine



FIG 186 Pedunculated adenoma (polyp) of the colon, removed by resection Early malignant change in one part of the tumour was found on microscopical examination

sigmoidoscopy performed because of coincidental ano-rectal disease such as hæmorrhoids, etc. Polyps at a higher level will usually be revealed by the barium enema, and the use of a reduction-density technique or air-inflation after evacuation of the barium are particularly useful for the detection of these small lesions. The finding of a polyp on sigmoidoscopy should indicate the necessity for X-ray examination of the colon, as another tumour, benign or malignant, is not infrequently present in the bowel. It is rarely possible to distinguish with certainty radiologically between a benign polyp of

the colon and an early carcinoma, and this must be remembered in planning treatment (Fig. 186).

Treatment. Pedunculated tumours within reach of the sigmoidoscope may be removed by endoscopic methods, such as the diathermy snare. Larger tumours and those situated more proximally in the colon require laparotomy. Until recently it was considered sufficient to remove polyps of the colon by colotomy; that is by opening the bowel over the tumour, removing it by ligaturing the pedicle, and then closing the bowel. Occasionally, however, subsequent examination revealed that malignant change had already occurred, and the prospects of final cure were thus doubtful. Furthermore, certain villous tumours have a more extensive distribution than is palpable through the bowel wall, and attempts at local excision via a colotomy incision are likely to result in incomplete removal. Improvements in chemotherapy, surgical technique, and anæsthesia have now made resection of the colon as safe a procedure as colotomy, and in most cases resection of a length of colon containing the tumour is now the operation to be preferred.

MULTIPLE POLYPOSIS

(Familial Intestinal Polyposis) (Adenomatosis)

THIS is a hereditary disease characterized by the development within the large bowel of large numbers of adenomatous tumours. It is a rare condition, which is passed on as a Mendelian dominant, usually by the marriage of a heterozygous affected person (Pp) with an unaffected individual (pp). Thus only half the offspring of the marriage are likely to inherit the disease (Fig. 187) Moreover, only the affected members can transmit

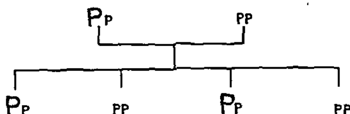
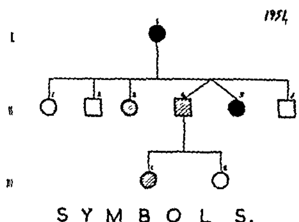


FIG 187 The inheritance of polyposis from one affected parent Only half the children are likely to be affected

it to their children; the descendants of unaffected members will be normal (Fig. 188). In a number of clinical cases of polyposis, careful inquiry into the family of the affected individual, including examination of relatives, has failed to reveal another member affected. Nonetheless, it seems that these solitary cases are manifestations of the same disease; it is likely that they represent the starting point of the disease in their family, and will transmit it to half their children. The severity of the disease appears to vary in different families, and in different individuals in a family. The tumours are not present at birth, but may develop in childhood, adolescence, or early adult life; in severe cases the mucosal surface of the bowel may be covered with innumerable tumours (Fig. 189). Microscopically, most of the tumours have the structure of adenomata, though some small villous tumours may also be present.

Carcinoma is almost certain to supervene in one or more of the adenomata in the rectum or colon at a relatively early age. The carcinoma that thus arises follows the normal course of large bowel carcinomata, and unless treated will lead to the death of the patient within a few years. This malignant change is the normal course and termination of the disease, but the rapidity with which the course is run varies widely. Thus several cases are known where malignancy has developed in the early twenties, and a few where a patient, undoubtedly affected and even transmitting the disease, has remained well without treatment into old age (Dukes, 1952).



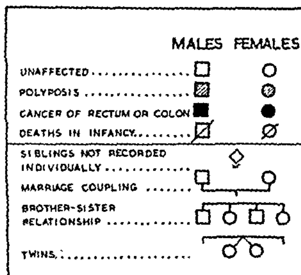


FIG 188 Family pedigree from an actual case of familial polyposis. (St Marks family 7) In each generation only half the children are affected

Clinical Features and Diagnosis. The symptoms of polyposis are often mild and their onset insidious. Usually there is some increased frequency of defæcation, with excess of mucus and sometimes blood in the motions. In severe cases both diarrhœa and bleeding may be marked, but there is no doubt that perfect health with freedom from all symptoms may be maintained even with numerous polypi.

Physical examination is usually entirely normal, and the diagnosis is made by sigmoidoscopy and X-rays, both of which are necessary in assessing the extent and severity of the disease; special techniques may have to be used by the radiologist to demonstrate the tumours when they are still small. The only condition likely to cause confusion in diagnosis is ulcerative colitis associated with "pseudo-polypi." In the latter the polyps are rarely so numerous, and on sigmoidoscopy the mucosa between them is seen to be red and granular. On X-ray, the bowel affected by colitis is often contracted with loss of haustration, while the outline remains normal in polyposis (Fig. 190).

Treatment

COLECTOMY WITH ILEO-RECTAL ANASTOMOSIS

This operation is probably the one of choice for most cases of polyposis. It is certainly not ideal, in that the mucosa of the rectum which is still likely to form polyps is retained, but the patient afterwards has full rectal control with only mild looseness and frequency of bowel action.

Before the operation is undertaken, the polyps must be cleared from the rectum (for about 12–15 cm. from the anus) by repeated diathermy coagulation through an operating

sigmoidoscope. When the rectum has been cleared, the operation is best done in one stage; the colon is excised down to the upper rectum, and the terminal ileum anastomosed end-to-end or side-to-end to the rectum.

Patients who have been treated in this way should attend at regular and frequent intervals afterwards for careful examination, and for early fulguration by diathermy of



FIG 189 Photograph of part of the colon removed from a patient with polyposis The tumours vary in size and shape.

any fresh rectal polyps that arise. If this is assiduously done, the risk of a carcinoma developing in the retained rectum is slight.

TOTAL PROCTO-COLECTOMY WITH TERMINAL ILEOSTOMY

This procedure becomes necessary in cases where the polypi in the rectum are so numerous that clearance of the rectal segment by diathermy is impossible without complete destruction of the mucosa, and in cases where carcinoma has already developed



(Mr. C. Naunton Morgan's case)

FIG 190. Barium enema (reduction-density technique) in a patient with polyposis Innumerable small tumours are present throughout the colon

in the rectum. The operation is best carried out in two stages, as in a case of ulcerative colitis (see p. 431), the first stage to consist of a colectomy from cæcum to mid-sigmoid, establishing a terminal ileostomy and a non-functioning sigmoid colostomy; and the second of an excision of the remaining rectal segment. The excision of the rectum may be done first if there is good reason, such as the presence of a carcinoma there, but otherwise it is better to establish only one functioning stoma, the ileostomy, to which the patient may get accustomed.

Procto-colectomy with Ileo-anal Anastomosis

Though theoretically ideal on pathological grounds, this operation deprives the patient of the sensory and reservoir functions of the rectum, and seldom results in satisfactory continence. The operation should be regarded as being still in the experimental stage.

It is not uncommon in cases of polyposis for several carcinomata to develop concurrently in the large bowel. A special search of the whole large bowel should therefore be made at laparotomy before deciding the extent of the resection.

Familial Intestinal Polyposis with Oral Pigmentation

Another very rare form of multiple intestinal polyposis of a hereditary nature has been described. In this syndrome, the polyps occur particularly in the small intestine, though the colon is usually also affected, and the patients tend to get repeated attacks of small bowel intussusception caused by a polyp; they have melanin pigmentation around the mouth, on the mucosa of the lips, and often on the buccal mucosa. The condition appears to be inherited in the same way as the more common polyposis of the large bowel (Wolff, 1952).

CARCINOMA OF THE COLON

Incidence and Site

Over 10,000 people in England and Wales die yearly from cancer of the colon, and the incidence of the disease is increasing slowly with an ageing population. Men and women are affected in nearly equal numbers, and the peak age of incidence is the sixth and seventh decades, though no age is exempt.

Fig. 191 shows in simple form the frequency with which various parts of the large bowel are affected by carcinoma. It will be seen that there are roughly as many growths in the rectum as there are in the entire colon, and that of the colonic growths about half occur in the sigmoid; the important corollary to this is that about two-thirds of all large bowel cancers are within reach of the finger or sigmoidoscope. In about 3 per cent of cases of large bowel cancer there is more than one primary growth.

Ætiological Factors

It is probable that many cancers of the bowel arise in *pre-existing benign tumours*, most of which have been symptomless. As evidence for this, there is the remarkable similarity between the distribution of polyps and that of cancer in the colon, and there

is the frequent finding of early cancerous changes in polyps removed surgically. Cases of *familial polyposis coli* nearly always develop cancers in the bowel, often multiple, and frequently at an early age. Recent observations have shown that cases of long-standing *ulcerative colitis* are liable to develop carcinomas of the rectum or colon: here again the cancers may be multiple, and they are commonly highly malignant.

Pathology

Carcinoma of the colon may take the form of a large protuberant mass, of a malignant ulcer with everted edges, or of a stenosing annular scirrhus growth. The protuberant

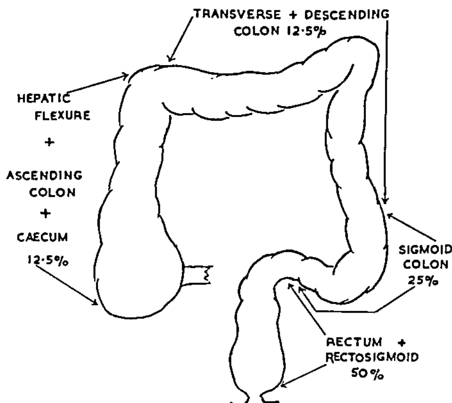


Fig 191 The distribution of carcinomas in the large bowel About half occur in the rectum, and half the remainder in the sigmoid

type of growth is most usual in the cæcum but is by no means confined to that region, and large bulky growths may be found in any part of the bowel. The small annular growths are rare and occur mainly in the left colon.

Microscopically, most of the growths are adeno-carcinomas, but vary in their degree of differentiation so that tumours of high, low, and intermediate grades of malignancy are encountered. Colloid carcinomas and anaplastic carcinomas account for the remainder. Anaplastic tumours and those of high malignancy metastasize early, so that the size of a growth is little indication of the presence or absence of metastases.

As in other carcinomas, spread occurs by direct infiltration, by lymphatic and venous embolism, and by seeding within the peritoneal cavity. The most important method of spread is by the lymphatics, and the extent of surgical excision must be planned so as to remove the widest possible lymphatic field, irrespective of whether the nodes are enlarged

or not; metastases may occur without enlargement of a node, or conversely an uninvolved node may be grossly enlarged as a result of sepsis. Metastases are frequently found in the liver from venous embolism, and rarely secondary deposits occur in the lungs. Direct spread may result in adherence to the abdominal parieties or to neighbouring viscera, and internal fistulæ are sometimes so caused. In this connection, it is important to realize that the highly infective nature of the content of the colon will lead to gross infection of any malignant ulcer. Thus the œdema and fibrosis of sepsis will increase both the bulk and fixity of the tumour, often to a considerable degree: size and fixation alone therefore, unless massive, should not be deemed to render a growth inoperable or the outlook hopeless. It is often found that very bulky adherent tumours are of a low grade of malignancy, and radical removal may often result in long survival.

Clinical Features

The symptoms of a colon cancer differ according to the site of the lesion. This is to be expected in view of the difference in calibre of the right and left colon, the different nature of the content in the two sides, and to a certain extent the nature of the growth, those on the right side being more frequently proliferating bulky growths.

The symptoms of growths in the *right half of the colon* are rarely directly suggestive of colonic disease. Pain is the most common symptom, but may be a diffuse abdominal ache or colic, or occur in the right iliac fossa and be ascribed to disease of the appendix. Sometimes nausea, dyspepsia, lack of appetite and loss of weight occur and simulate upper abdominal disease. Not infrequently, fatigue and weakness due to anæmia may be the presenting symptoms, and such patients may be treated for secondary anæmia for some time before the underlying cause is discovered. An alteration in bowel habit, with either constipation or diarrhœa, and sometimes periods of both, occur in only about 30 per cent of these patients, while in only about 10 per cent is blood noticed in the motions. In a small number of cases the finding of a lump in the right iliac fossa, either by the patient or the doctor, may be the first indication of disease. Such a mass is palpable on examination in about two-thirds of all patients with carcinoma of the right half of the colon, and should always be carefully sought (Spear and Brainard, 1951, Costello, 1952).

In cancer of the *left half of the colon*, where the content of the colon is solid, obstructive symptoms usually predominate. Again pain is probably the earliest and most frequent symptom, and is at first a rather vague recurrent aching or colic in the lower abdomen. As obstruction increases, the severity and frequency of the attacks of colic will increase, and may culminate in a complete intestinal obstruction. Alteration in bowel habit is very common, constipation is the more usual, but attacks of diarrhœa are likely to occur, and more frequently cause the patient to seek medical advice. Blood in the stools is noticed by the patient in about 50 per cent of cases. In less than a quarter of left-sided growths will a mass be palpable on examination, either in the abdomen or through the rectal wall.

A certain number of cases of cancer of the colon come under medical care for the first time when they develop acute intestinal obstruction. The percentage of cases so presenting has varied from 5 to 25 per cent in different series published, but in about 90 per cent of cases the site of the obstructing carcinoma was in the left colon. It is rare for a carcinoma of the right colon to lead to complete obstruction until a very late stage.

is the frequent finding of early cancerous changes in polyps removed surgically. Cases of *familial polyposis coli* nearly always develop cancers in the bowel, often multiple, and frequently at an early age. Recent observations have shown that cases of long-standing *ulcerative colitis* are liable to develop carcinomas of the rectum or colon: here again the cancers may be multiple, and they are commonly highly malignant.

Pathology

Carcinoma of the colon may take the form of a large protuberant mass, of a malignant ulcer with everted edges, or of a stenosing annular scirrhus growth. The protuberant

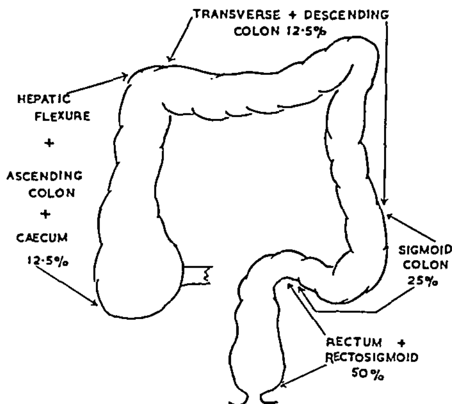


FIG 191. The distribution of carcinomas in the large bowel About half occur in the rectum, and half the remainder in the sigmoid

type of growth is most usual in the cæcum but is by no means confined to that region, and large bulky growths may be found in any part of the bowel. The small annular growths are rare and occur mainly in the left colon.

Microscopically, most of the growths are adeno-carcinomas, but vary in their degree of differentiation so that tumours of high, low, and intermediate grades of malignancy are encountered. Colloid carcinomas and anaplastic carcinomas account for the remainder. Anaplastic tumours and those of high malignancy metastasize early, so that the size of a growth is little indication of the presence or absence of metastases.

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(Courtesy of Dr. Norman Henderson)

FIG 192 Barium enema—"filling-defect" due to a carcinoma of the lower sigmoid colon. Note the abrupt margins, and the irregular outline of the defect.

In a very few cases, perforation of the bowel either through or just above the growth and causing peritonitis first brings the patient to hospital as an emergency.

Diagnosis

If the results of the surgery of colon cancer are to improve, it is essential that cases should reach the surgeon at an earlier stage: the diagnosis must be made or suspected sooner. It is important that doctors should be familiar with the possible early symptoms, and should arrange for investigation when suspicion is aroused. Thus any bleeding from the bowel, any change of bowel habit, any abdominal colic, any pain or lump in the right iliac fossa, any unexplained and persisting anæmia, indigestion, or ill-health, should all suggest the possibility of a colon cancer, and call for investigation. Even one attack of diarrhœa in a patient over 40 requires investigation unless there has been an obvious infective cause.

In confirming the diagnosis, sigmoidoscopy and X-rays may both be necessary and should be employed in that order. The sigmoidoscope will reveal growths in the lowermost 10 or 12 in. of the bowel, or by demonstrating blood in the lumen may indicate a lesion at a higher level. Barium enema undertaken by a skilled radiologist will usually demonstrate a carcinoma as a "filling-defect" in the outline of the bowel (Fig. 196), but oblique as well as antero-posterior views must be taken; the barium enema must not be relied on to demonstrate lesions in the upper rectum and recto-sigmoid, where sigmoidoscopy is essential. Further, negative results from investigation on one occasion do not exclude the presence of a small tumour, benign or malignant, in the colon, and investigations should be repeated if symptoms persist. Occasionally, exploratory laparotomy is indicated by the finding of blood on sigmoidoscopy or continued clinical *suspicion, despite a completely normal barium enema.*

Differential Diagnosis

Diagnoses such as "piles" and "colitis" can be excluded by proper examination. The condition which most closely simulates carcinoma both clinically and radiologically is diverticulitis of the sigmoid, and distinction between them is sometimes impossible (see p. 418); they may both, of course, be present concurrently. Benign tumours of the colon are another source of error, but the treatment is on similar lines to carcinoma so the distinction is less important.

A palpable carcinoma of the colon must be distinguished both from other lesions affecting the bowel and from swellings of other viscera, such as the kidneys, liver, gall bladder, or spleen Carcinoma of the cæcum in particular needs to be distinguished from an appendix mass, Crohn's disease, actinomycosis, and hyperplastic tuberculosis, and *the diagnosis is made largely from the history and clinical signs, as deformity of the* cæcum on barium enema may be seen in all. The possibility of amœbic granuloma should also be borne in mind. In any part of the colon a regional colitis may cause a clinical and radiological picture very similar to that of carcinoma.

Treatment

Principles. The aim of surgical treatment is the removal of the growth with as wide a margin as possible, both locally and in the lymphatic field, with restoration of normal continuity and bowel function.

test of blood-supply is to see fresh arterial bleeding from the cut edges of both ends, which should be sectioned obliquely so as to ensure an ample blood-supply to the anti-mesenteric side. An open anastomosis in two layers without clamps is simple and safe, and there is no advantage in closed aseptic anastomosis; nor is there any necessity to drain an intraperitoneal anastomosis unless there has been considerable soiling. If there is the slightest doubt as to the perfection of the anastomosis, a temporary proximal colostomy diverting the fæcal stream is advisable.

There is still a very limited place for staged extra-peritoneal colectomy (the Paul-Mikulicz operation) in the surgery of colon cancer. It might be considered in the following circumstances:

(1) Cases when in spite of preparation an unexpected degree of loading of the bowel is found to persist at operation, and delay in order to do a preliminary colostomy is not considered advisable. Primary anastomosis under these conditions may be hazardous.

(2) Cases when the patient's condition deteriorates during the operation, which must be completed rapidly.

It must be emphasized that the extent of the resection in the lymphatic field in an extra-peritoneal colectomy should be as radical as by any other technique, and thus the method should not be used for growths in the sigmoid colon (*see* p. 450). Though advocated by some for right hemi-colectomy, it has never been widely adopted for this operation, in which anastomosis has always been preferred.

Cancer in Different Sites

Cæcum, Ascending Colon, Hepatic Flexure

Unlike growths of other regions of the large bowel, cæcal cancers are slightly more common in women than in men. Frequently bulky, they may be fixed to the abdominal parietes, but seldom invade any organ which cannot be removed. Growths in the region of the hepatic flexure, however, may become adherent to the liver, to the gall bladder, or to the second part of the duodenum and head of pancreas. Though such adherence may make the operation more difficult and hazardous, a radical removal of the neoplasm and all attached structures should be attempted in the absence of liver metastases or other dissemination. Careful exploration and assessment is needed before the removal is started, for adherence to the superior mesenteric vessels or to the portal vein and common bile duct usually render the growth inoperable. The lymphatic drainage from these growths is to glands lying along the ileo-colic and right colic vessels, which should be tied at their origin from the main superior mesenteric trunk (Fig. 198). The right branch of the middle colic should be tied high for growths at the hepatic flexure and it is sometimes advisable to tie the main trunk of that vessel. Hence a right hemi-colectomy is always necessary, and continuity must be restored by anastomosis between lower ileum and transverse colon. End-to-end anastomosis is often the simplest method, but if there is considerable difference in calibre between ileum and colon, end-to-side anastomosis with closure of the end of the colon is preferable—the blind end of colon should be as short as possible. It is not necessary to reperitonealize the right colic gutter, but the gap in the mesentery must be closed.

Cases presenting with acute obstruction usually require a staged procedure. A preliminary ileo-transverse anastomosis in continuity relieves the obstruction, and resection is undertaken 2–3 weeks later. Occasionally the ileo-cæcal valve remains

Preparation

The patient should come to operation in the best possible general condition, and with any anæmia, protein or vitamin deficiency corrected. Admission to hospital well before the operation is advisable in order to ensure this.

Ideally the colon should be so prepared that at operation it is collapsed and empty and its contents sterile. Though this ideal is seldom fully attainable, satisfactory operating conditions can often be acheived, even in patients with considerable obstruction, by means of mild aperients, bowel washouts, and a low-residue diet. For the last few days before operation insoluble sulphonamides and antibiotics should be given to sterilize the contents of the bowel (Lockhart-Mummery, 1954).

Patients presenting with acute obstruction require an operation to relieve the obstruction, usually a colostomy (*see* p. 294). No attempt should be made to remove the growth at this stage, as a fully radical operation can hardly ever be achieved in the presence of obstruction, and the risks of attempting it are great. Two or three weeks later, when the distension and œdema will have settled and the bowel has been prepared, removal of the primary growth can be attempted. A few patients with severe chronic obstruction will be seen who are best treated on similar lines with a preliminary colostomy, but most patients can be satisfactorily prepared for resection in one stage as outlined above.

Essentials of Operation

Good relaxation and an adequate well-placed incision are essential; a long right or left paramedian incision will be found to give good access in most cases. It has already been emphasized that local fixity seldom prevents removal; when possible, other organs to which the growth has become adherent should be removed *en bloc* without attempting separation. Wide mobilization of the colon above and below the growth is the essential step in the surgery of the large bowel; the meso-colon and its vessels are thus displayed, and it is often surprising how wide an area of bowel and mesentery can be removed and still allow the two ends to come together without tension.

Choice of Method

In the early days of abdominal surgery, the resection of part of the colon and restoration of continuity by anastomosis carried a formidable mortality. Between 1890 and 1900, Bloch of Copenhagen, Paul of Liverpool, and Mikulicz of Breslau all described, apparently independently, a staged extra-peritoneal method of resection of the colon, which greatly reduced the risk of peritonitis. This method was adopted with minor modifications in technique throughout the world, and enormously improved the results and decreased the mortality of the surgery of colon cancer. Advances in anæsthesia and surgical technique, and the advent of the sulphonamides and antibiotics, however, have greatly increased the safety of intestinal surgery, and there has been a tendency in the last decade to abandon the staged extra-peritoneal methods in favour of primary anastomosis. This is not only more pleasant for the patient, but more important, it often permits a more radical cancer operation to be carried out. Experience has shown that operative mortality is no higher provided that certain principles are observed.

Safe anastomosis requires that both ends of bowel to be joined should have a good blood supply, and that they should come together without tension. The only reliable

The operation may be completed by immediate end-to-end anastomosis, or by a double-barrelled colostomy for later closure (Paul-Mikulicz method), depending on the particular circumstances and the surgeon's wish. Cases of acute obstruction due to a growth in the transverse colon are best treated by cæcostomy or by a diversionary colos-

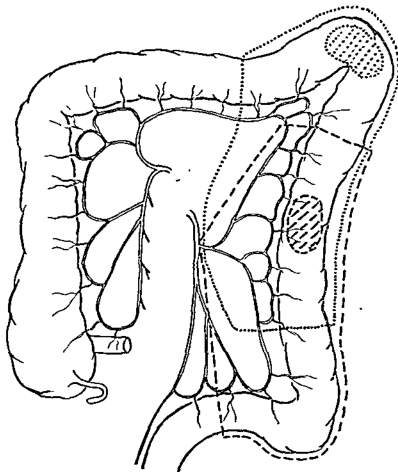


Fig 194 Extent of resection necessary for growths at the splenic flexure and in the descending colon.
Carcinoma of Splenic Flexure
- - - - - Carcinoma of Descending Colon

tomy in the ascending colon. A colostomy in the transverse colon proximal to the growth would be a hindrance to a wide resection at the later operation, and is best avoided.

Splenic Flexure and Descending Colon

Growths in the region of the splenic flexure are often relatively "silent" and are frequently advanced before the diagnosis is made. Acute obstruction often first brings the patient under medical care. The growth if large may become adherent to the stomach, the spleen or the left kidney, and these organs may also require removal. Lymphatic drainage is both to glands along the left colic artery and to a lesser extent towards the origin of the middle colic artery. In dealing with growths at the splenic flexure it is necessary to ligate the left colic at its origin and to remove omentum and mesentery widely on the proximal side. For growths in the descending colon the first sigmoid artery may also need to be taken (Fig. 198).

competent, so that the cæcum becomes very tense while the lower ileum is little dilated; in such cases a cæcostomy is essential to prevent rupture, or alternatively immediate resection and anastomosis after aspiration may be justified.

Transverse Colon

Growths of this part of the bowel are often large, and in many cases a mass will be palpable at the first abdominal examination. If the growth spreads through the bowel wall, the stomach is the organ most frequently adherent, and a gastro-colic fistula is sometimes caused This is usually no bar to operative removal, as partial gastrectomy can easily be carried out too, but adherence posteriorly to the body of the pancreas or vital vessels is less favourable and often indicates inoperability.

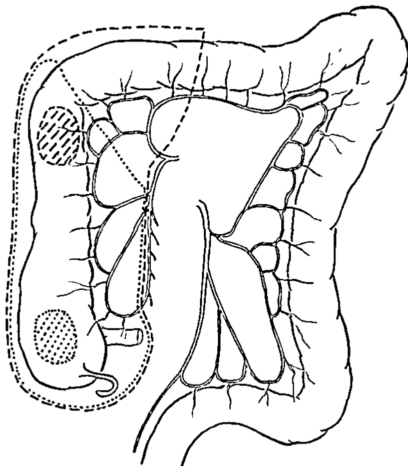


Fig 193 Extent of resection necessary for carcinomas in the right colon For growths at the hepatic flexure, the main trunk of the middle colic artery may need to be taken
Carcinoma of the Cæcum
- - - - - Carcinoma of the Hepatic Flexure

The lymphatic drainage from most of the transverse colon is to glands along the middle colic artery, which should therefore be tied as near to its origin from the superior mesenteric as possible, and a wide extent of mesentery should be taken. It will often be necessary to mobilize both flexures in order to allow an adequate resection and anastomosis without tension, and most if not all of the great omentum and gastro-colic omentum should be removed with the growth.

become adherent to uterus, rectum, or bladder sometimes causing a vesico-colic fistula. Partial cystectomy and hysterectomy may therefore be needed, and if necessary it is usually possible to remove most of the rectum in continuity with the sigmoid and still restore continuity; rarely this is not possible and a combined excision of the rectum becomes necessary. Advanced growths at or below the pelvic brim may become adherent to the ureter or iliac vessels, but can usually be freed by careful dissection.

Lymphatic drainage is to glands along the sigmoid and superior hæmorrhoidal vessels, and then further to glands round the inferior mesenteric trunk. A wedge resec-

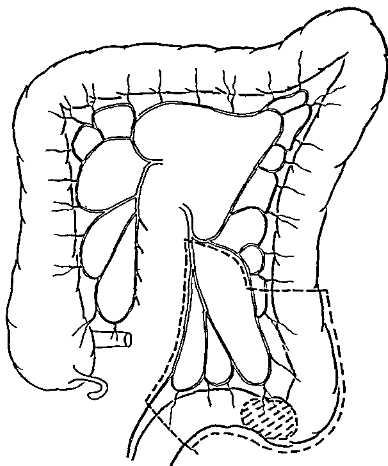


FIG. 196 Extent of resection necessary for growths of the sigmoid colon The inferior mesenteric artery is tied just below the origin of the left colic artery

tion of part of the sigmoid colon with ligature of the sigmoid vessels where they arise from the superior hæmorrhoidal is no longer considered adequate. In every sigmoid growth the main superior hæmorrhoidal vessels should be tied just below the origin of the left colic, with anastomosis of descending colon to upper rectum (Fig. 196). The blood supply to the rectum is not arrested by ligature of the main superior hæmorrhoidal vessels as was formerly believed, the inferior and middle hæmorrhoidals alone being entirely adequate to supply the bowel certainly as high as the sacral promontory and probably higher. In certain cases, a more radical operation can be carried out by ligature of the main inferior mesenteric trunk, as has already been discussed in the previous section. Though this will often require anastomosis between left transverse colon and

Operations for cancer on the left side of the colon can be made more extensive and radical by ligating the inferior mesenteric trunk as it arises from the aorta, thus removing a wider extent of mesentery and the lymphatic field to a higher level. The left extremity of the transverse colon can then be swung down and joined to the upper rectum to restore continuity (Fig. 195). This operation should certainly be done for growths with extensive

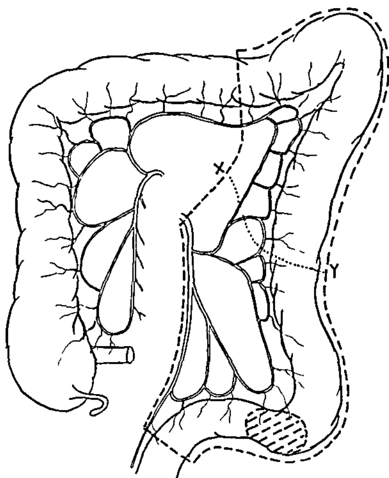


Fig 195 More extensive resection for growths in the left colon. The inferior mesenteric artery is tied at its origin from the aorta Following this, the blood supply through the marginal artery is often sufficient to nourish most of the left colon, and the upper extent of the resection may then be along the line X-Y, which is equally radical lymphatic clearance for growths of the sigmoid colon

lymphatic involvement, but whether it is advisable for all growths in the left colon is still debatable. The technical difficulties are somewhat greater, particularly in fat subjects, with a corresponding increase in the dangers over those of the standard operation.

Cases of acute obstruction due to growths in the descending colon are best treated by a colostomy in the right half of the transverse colon. Cæcostomy, or colostomy in the ascending colon are probably better for growths at the splenic flexure, as the whole transverse colon is then left free for mobilization if necessary.

Sigmoid Colon

This is the most frequent site of colon cancer. The sigmoid loop frequently lies in the recto-vesical or recto-uterine pouch, and growths arising in this part of the colon may

become adherent to uterus, rectum, or bladder sometimes causing a vesico-colic fistula. Partial cystectomy and hysterectomy may therefore be needed, and if necessary it is usually possible to remove most of the rectum in continuity with the sigmoid and still restore continuity; rarely this is not possible and a combined excision of the rectum becomes necessary. Advanced growths at or below the pelvic brim may become adherent to the ureter or iliac vessels, but can usually be freed by careful dissection.

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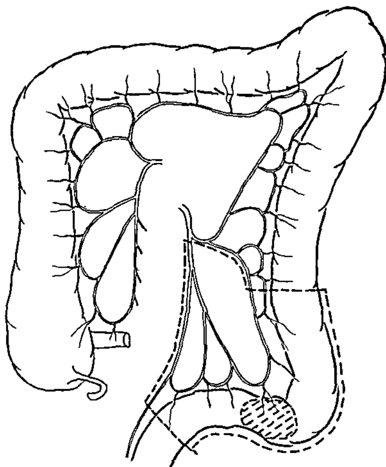


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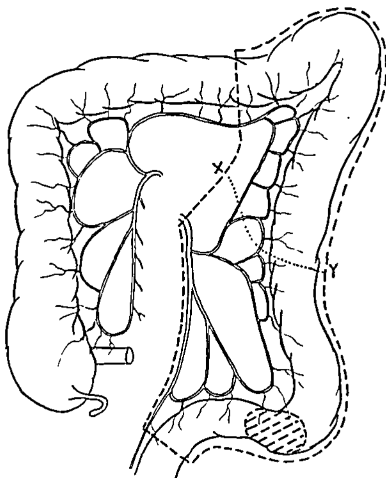


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COLOSTOMY—CÆCOSTOMY—ILEOSTOMY

Colostomy

A COLOSTOMY may be terminal, when the distal bowel has been excised, or of loop type in continuity, or the bowel may be divided and both ends brought out separately. It is possible, after mobilization, to establish a colostomy in any part of the colon, but the sigmoid colon or transverse colon are the usual sites. The more distally in the bowel the stoma is made, the more formed will be the motion.

Management. Full control cannot be obtained with a colostomy, because neither the sensory arc nor the sphincter muscles that together make for rectal continence can be duplicated. However, in most cases the bowel will acquire its own rhythm within a few weeks, and will act at regular times each day, usually twice in 24 hours. The patient will soon learn what stimuli (such as a morning cup of tea) will provoke an action, and will also learn what articles of diet are to be avoided as likely to cause looseness and irregularity. By attention to this natural rhythm and to their diet, patients are thus able to exercise a considerable measure of control over their colostomies and lead normal lives with minimal disability (Gabriel, 1949, Dukes, 1947).

Certain patients never attain a regular rhythm, and may have frequent small actions which make the colostomy a burden. These patients are happier with regular self-administered washouts of the bowel, and should be taught the technique. The essential points are that the catheter used must have a soft blunt tip, that force should never be used when inserting it into the bowel, that tap-water should be used for the washout, and that the fluid should be run in at low pressure from a can. The only danger of the washout regime is perforation of the bowel, which will be prevented by attention to these points.

A patient with a colostomy should wear a pad of gauze and cellulose over the stoma, and a wide firm belt to give some support to the abdominal wall. A flat disc of plastic or cardboard may be incorporated in the dressings over the stoma, but cup appliances should never be used as they invariably lead to hernia formation by suction.

Complications. The most common complications are *stenosis* at skin level, and *ventral hernia* round the colostomy; minor degrees are frequent, and if severe they may require reconstruction of the stoma. Loop colostomies are more liable to the complication of *prolapse*, particularly in children, while sometimes the opposite complication, *retraction of the spur*, necessitates refashioning. *Small gut obstruction* may occur round any artificial stoma, but is particularly likely to happen if a loop of bowel passes down between a left iliac colostomy and the left flank. This can be prevented by closing this lateral space with a purse-string suture when making the colostomy.

Cæcostomy

Cæcostomy is still sometimes performed in cases of obstruction of the large bowel. The cæcum may be mobilized and exteriorized over a rod, as in making a loop colostomy, but more frequently a large-size catheter is sutured into the cæcum which is then fixed to the anterior parietal peritoneum at the wound margins. This type of tube cæcostomy requires repeated gentle irrigation to keep the tube patent and prevent blockage, but if this is done it is very effective in decompressing the colon. It has the advantage that closure is usually spontaneous and fairly rapid when the tube is removed.

rectum, in many cases the blood-supply from the middle colic through the marginal artery is ample to the whole descending colon, allowing an equally radical operation with less mobilization of bowel (Fig. 196).

It will be appreciated that these more radical operations require restoration of continuity by intra-abdominal anastomosis, as after ligation of the superior hæmorrhoidal artery the blood-supply to the distal sigmoid is inadequate, although the rectum is well nourished; so there is not enough distal bowel to reach the surface and allow of an extraperitoneal anastomosis.

Acute obstruction from a growth of the sigmoid colon is best dealt with by a colostomy in the right side of the transverse colon, where it will not interfere with the later operation. The temptation to do an immediate Paul's operation for an obstructing growth in the sigmoid should be resisted; clearance both in bowel and mesentery is seldom adequate, and local recurrence is not infrequent.

Palliative Procedures. Cases will be seen which are beyond surgical cure, either because of clinical or radiological evidence of metastases, or because metastases are found when the abdomen is explored. If, as is often the case, the primary growth is still locally operable, it is usually advisable to resect it with immediate anastomosis as this *often seems to delay the progress of the disease as well as preventing obstruction. In* cases where the growth is fixed and removal would be hazardous, a palliative short-circuit is often possible and obviates the risk of obstruction. Ileo-colostomy for growths of the right colon and colo-colostomy between transverse colon and sigmoid for growths of the left colon are useful procedures; side-to-side anastomosis in continuity is safest, as this method avoids any long blind loops of gut which might later cause trouble.

It has already been explained that local fixity is seldom a sound reason for declaring a growth inoperable. With no other cancer is courageous surgery more amply rewarded, and patients with large and apparently fixed growths should never be declared inoperable without a laparotomy and often a trial dissection.

Results

The advances in medical science in the last decade are reflected in this branch of surgery as in many others. Thus the operative mortality of resections of the colon for cancer as published recently by several authors has been under 4 per cent, while the operability rate (the removal of the primary tumour) is over 90 per cent (Colcock, 1951, Morgan, 1952).

The end results naturally depend on the histological grade of malignancy of the tumour and on the extent of its spread at the time of removal. Of all patients submitted to radical operation in the absence of hepatic or other irremovable metastases, about 50 per cent are alive at 5 years. The results are slightly better for growths of the right colon than for those of the left side.

Other Malignant Tumours

Sarcomas of the colon are very rare. Leiomyosarcomas have been reported, as also have lymphosarcomas and other forms of the reticuloses. *Malignant melanomas* are equally uncommon. *Carcinoid tumours* are rare in the colon, and most have been accidental findings in specimens removed for other reasons; a few cases have been reported which presented as obstructive lesions and were found to have lymphatic metastases.

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Management. Full control cannot be obtained with a colostomy, because neither the sensory arc nor the sphincter muscles that together make for rectal continence can be duplicated. However, in most cases the bowel will acquire its own rhythm within a few weeks, and will act at regular times each day, usually twice in 24 hours. The patient will soon learn what stimuli (such as a morning cup of tea) will provoke an action, and will also learn what articles of diet are to be avoided as likely to cause looseness and irregularity. By attention to this natural rhythm and to their diet, patients are thus able to exercise a considerable measure of control over their colostomies and lead normal lives with minimal disability (Gabriel, 1949, Dukes, 1947).

Certain patients never attain a regular rhythm, and may have frequent small actions which make the colostomy a burden. These patients are happier with regular self-administered washouts of the bowel, and should be taught the technique. The essential points are that the catheter used must have a soft blunt tip, that force should never be used when inserting it into the bowel, that tap-water should be used for the washout, and that the fluid should be run in at low pressure from a can. The only danger of the washout regime is perforation of the bowel, which will be prevented by attention to these points

A patient with a colostomy should wear a pad of gauze and cellulose over the stoma, and a wide firm belt to give some support to the abdominal wall. A flat disc of plastic or cardboard may be incorporated in the dressings over the stoma, but cup appliances should never be used as they invariably lead to hernia formation by suction.

Complications. The most common complications are *stenosis* at skin level, and *ventral hernia* round the colostomy; minor degrees are frequent, and if severe they may require reconstruction of the stoma. Loop colostomies are more liable to the complication of *prolapse*, particularly in children, while sometimes the opposite complication, *retraction of the spur*, necessitates refashioning. *Small gut obstruction* may occur round any artificial stoma, but is particularly likely to happen if a loop of bowel passes down between a left iliac colostomy and the left flank. This can be prevented by closing this lateral space with a purse-string suture when making the colostomy.

Cæcostomy

Cæcostomy is still sometimes performed in cases of obstruction of the large bowel. The cæcum may be mobilized and exteriorized over a rod, as in making a loop colostomy, but more frequently a large-size catheter is sutured into the cæcum which is then fixed to the anterior parietal peritoneum at the wound margins. This type of tube cæcostomy requires repeated gentle irrigation to keep the tube patent and prevent blockage, but if this is done it is very effective in decompressing the colon. It has the advantage that closure is usually spontaneous and fairly rapid when the tube is removed.

Ileostomy

Apart from the rare occasion when a loop ileostomy may need to be established as an emergency, a terminal ileostomy is preferable; that is the bowel is divided, and the proximal end brought out through a stab incision as the functioning stoma. The distal end may be oversewn and dropped back, or exteriorized well away from the functioning ileostomy, or removed with the colon. It is important that the stoma should be away from bony prominences and from the umbilicus to allow of proper fitting of an appliance.

Management. An ileostomy differs from a colostomy in that it acts at frequent and less regular intervals, and that the efflux is more fluid; hence some form of appliance is necessary in which the efflux can be collected. Many types have been designed, and there is no doubt that appliances which adhere to the skin round the stoma are the most satisfactory. The original appliance of this design was the American Koenig-Rutzen bag, but other forms, adhering either by a special paste or by double-sided zinc oxide plaster are now available in this country. With such an appliance, the patient is able to return to work and to pursue all normal social activities. A good ileostomy should cause the patient very little trouble, and there is no doubt that an ileostomy is compatible with perfect health (Counsell and Lockhart-Mummery, 1954).

Complications

"Ileostomy Dysfunction"

This name is given to the partial intestinal obstruction which is the most frequent complication after ileostomy, occurring in some degree in about half the patients. It is due partly to the fixation of the lower ileum and partly to the resistance to intestinal flow caused by the abdominal wall, and is accentuated by any scarring or contraction at the stoma. It is characterized by mild colic, irregularity of ileostomy action—sometimes complete cessation but more usually a persistently profuse and liquid efflux—and sometimes by vomiting and fluid and electrolyte imbalance, the latter often severe. It occurs most frequently in the second week after ileostomy, and the patient may become ill and toxic and lose weight as a result (Warren and McKittrick, 1951).

Treatment in mild cases is by digital dilation of the stoma and the frequent passage of a catheter into the ileum; in more severe cases the ileostomy may need to be reconstructed.

Prolapse

Some patients seem to be particularly prone to this complication, which is not always prevented by fixing the ileal mesentery to the abdominal wall when making the ileostomy. If the prolapse is only an inch or two no treatment is necessary. However, a massive prolapse may occur and even strangulate; in such cases operation is required and the ileostomy must be refashioned. Unfortunately there can be no guarantee that a similar course of events will not happen again.

Retraction

The ileostomy may retract to skin level when the patient lies down, and this leads to difficulty in management. The ileostomy should be reconstructed if stenosis has occurred or if the retraction is making control by the appliance difficult.

Fistula

This most often occurs at skin level, and is due to trauma, the responsible agent being in most cases the sharp rim of an appliance which has slipped and rubbed against the ileal spout. It may also arise from rough handling or careless suturing at the time that the ileostomy is made.

Sometimes joining the fistula and the main opening will restore a satisfactory ileostomy, but in many cases it is necessary to reconstruct the ileostomy and amputate the last inch or so of ileum which includes the fistula.

References

Congenital Abnormalities

Dott (1923) *Brit. J. Surg.* 11, 251.
Evans, A. (1929) *Brit. J. Surg.* 17, 34.

Megacolon

Bodian, M., Stephens, F. D. and Ward, B. C. H. (1949) *Lancet*, 1, 6.
Williams, D. I. (1954) *Annals R.C.S. of England*, 14, 107.
Maunsell, H. W. (1892) *Lancet*, 2, 473.
Weir, R. F. (1901) *J. Amer. Med. Ass.* 37, 801.
Swenson, O. and Bill, A. H. (1948) *Surgery*, 24, 212
Swenson, O. (1950) *Surgery*, 28, 371.
Swenson, O. (1951) *Pediatrics*, 8, 542
Browne, Denis. (1949) *Proc. Royal Soc. Med.* 42, 227.
Bodian, M., Carter, C. O. and Ward, B. C. H. (1951) *Lancet*, 1, 302.
Bodian in Fanconi and Wallgren's Textbook of Paedriatics. *Heinemann*, 1952.

Volvulus

Bruusgaard, C. (1947) *Surgery*, 22, 466.
Lyall, A. (1946) *Brit. J. Surg.* 33, 295.

Amœbiasis

Theron, P. (1947) *Brit. Med. J.* 2, 123

Diverticulosis and Diverticulitis

Edwards, H. (1939) Diverticula and Diverticulitis of the Intestine. *Wright and Son*, Bristol.
Devine, H (1938) *Surgery*, 3, 165
Lloyd-Davies, O V. (1953) *Proc. Royal Soc Med* 46, 407.

Ulcerative Colitis

Sammons, H. G. (1951) *Lancet*, 2, 239
Wells, C. (1952) *Annals R.C S. of England*, 11, 105.
Counsell [illegible]
[illegible] 14.
Corbett, R S (1952) *Annals R C S. of England*, 10, 21.
Gabriel, W. B (1952) *Brit Med. J.* 1, 881
Aylett, S. O. (1953) *Brit Med. J.* 2, 1348.
Rice-Oxley, J M. and Truelove, S. C (1950) *Lancet*, 1, 663.

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CHAPTER XV

ABDOMINAL ACTINOMYCOSIS

ZACHARY COPE

ABDOMINAL actinomycosis is not common but it occurs more frequently than is generally supposed. Many cases are missed during life and discovered only at post-mortem examination.

Actinomyces bovis often lies latent in the crevices of carious teeth and portions of the organism may be swallowed. If they survive they may give rise to pathological lesions in the wall of, but more often outside and adjacent to the particular part of the intestinal canal in which they happen to be. The varieties which occur are gastric, intestinal, ileocæcal, and colonic; from any of these the liver may be involved by metastasis.

Gastric Actinomycosis. The actinomyces may actually form an inflammatory mass in the wall of the stomach. Behring described such a lesion which caused pyloric stenosis; partial gastrectomy was performed and the actinomyces was identified in microscopic sections of the pylorus (*see* Fig. 1). More commonly the actinomycotic lesion develops after perforation of a peptic ulcer; one assumes that the organism escapes at the time of perforation and slowly develops outside the stomach. Mr. Edric Wilson has given me the clinical history of such a case.



(*From "Actinomycosis," Zachary Cope, Oxford University Press*)

FIG 197 Actinomycosis of duodenum. (After Behring)

A woman aged 54 was admitted to hospital with three days history of colicky right upper quadrant pain, vomiting, flatulence, and pyrexia. The signs and symptoms were those of acute cholecystitis. The attack subsided under treatment by sulphadiazine for ten days.

Two months later the patient was re-admitted to hospital She had lost two stones in weight and [illegible] pyrexia. Barium meal showed the [illegible] almost complete achlorhydria. [illegible] pus which escaped contained [illegible] complete disappearance of the mass.

It would appear likely that the initial attack was due to a small perforation of the pyloric part of the stomach.

With patients in whom a perforated peptic ulcer has been successfully sutured but whose post-operative course is not satisfactory, the surgeon must always consider the possibility of a sub-hepatic focus of actinomycosis. In most cases the signs may point to a sub-phrenic abscess and diagnosis will depend upon detecting the organism in the pus.

Carcinoma of the Colon

Spear, H. C. and Brainard, S. C. (1951) *Annals Surg.* **134**, 934.
Costello, C (1952) *Cancer*, **5**, 254.
[illegible] 30, 192
[illegible]
[illegible] ic (*W. B. Saunders Co;* Philadelphia).

Colostomy, Cæcostomy, Ileostomy

Gabriel, W. B. (1949) Principles and Practice of Rectal Surgery. *Fourth edition. H. K. Lewis and Co.*, London.
Dukes, C. E. (1947) *Lancet*, **2**, 12.
Counsell, P. B. and Lockhart-Mummery, H. E. (1954) *Lancet*, **1**, 113.
Warren, S and McKittrick, L. S. (1951) *Surg. Gynec. Obstet.* **93**, 555.

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Actinomycosis of the small intestine is very rare. In the remarkable case recorded by Sir G. Gordon-Taylor a large hard mass adjacent to the small bowel closely simulated a malignant mass and was successfully excised together with the affected bowel (*see* Fig. 177).

Ileo-cæcal Actinomycosis. The most common actinomycotic lesion within the abdomen is that which arises in the right iliac fossa following an attack of acute perforating appendicitis. Presumably the organism escapes from the lumen of the appendix at the time of perforation. Nothing unusual is noted at the time of operation, but within two or



FIG 198 X-ray of thorax and abdomen after infection of iliac sinus (Sir Heneage Ogilvie's case)

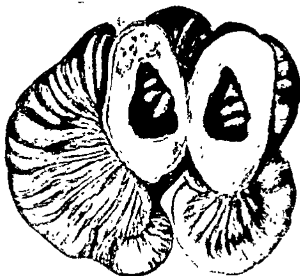


FIG 199 Actinomycotic mass in mesentery of small intestine (Sir G Gordon-Taylor)

(From "Actinomycosis," Zachary Cope, Oxford University Press)

three weeks a hard lump develops in the right iliac fossa and around the sinus (if a drainage tube has been inserted). The mass is painless and, unless correctly diagnosed and treated, steadily increases in size. It may spread upward toward the kidney and liver, medially through the psoas towards the spinal column, or downwards into the pelvis. If a sinus has persisted it may be possible to obtain the actinomyces from it. Otherwise, sooner or later, an abscess will form and come to the surface, where it will be incised and reveal the causative organism.

The hardness and painlessness of the mass may simulate malignant disease, but the low irregular fever, gradual extension, and the fact that it follows an operation for appendicitis, generally put one on the right track.

Sometimes a similar lesion arises without the history of any recent attack of appendicitis. One must assume that there had been an attack which was mild and unnoticed. The diagnosis is correspondingly more difficult until a local abscess forms

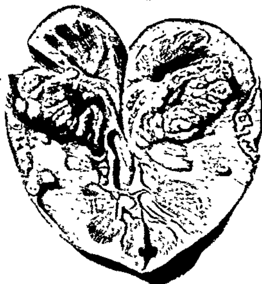
The insidious way in which the disease may spread from the iliac fossa was well shown in the case of a patient who was under the care of Sir Heneage Ogilvie;

...the upper part of this track was continuous with the anterior
was outlined. Treatment by penicillin
nce of lump (*see* Fig. 3).

Actinomycosis of the Colon. Actinomycosis of the colon has always (so far) been mistaken for cancer of the colon until very late in the course of the disease. It has usually been diagnosed by the examination of a microscopic section of the excised mass, or by the finding of the actinomyces in the pus of an abscess which has formed and pointed on the surface.

Actinomycosis of the Liver is not uncommon. It may be secondary to a focus in the stomach, intestine or elsewhere within the portal area, or it may be due to direct extension from an ileo-cæcal mass. At first the lesion in the liver is firm in texture and, to naked eye inspection, looks like a focus of malignant disease. Later it softens, forms loculated abscesses, and may by extension infiltrate the abdominal wall and form a superficial abscess. Alternatively the suppurative process may invade the right lobe of the diaphragm and rupture into the thorax, either causing an empyema or infecting the bronchial tract.

Occasionally the primary focus of actinomycosis may have been very small and insignificant and may not have caused any symptoms; in such a case the hepatic focus may appear to be primary and cause great difficulty in diagnosis. It may need an abdominal exploration and the removal of a small portion for biopsy in order to make certain of the diagnosis.



(From "Actinomycosis," Zachary Cope, Oxford University Press)
FIG. 200 Solitary actinomycosis of kidney (Israel)

The possibility of actinomycosis must always be considered when irregular swellings occur in the liver, particularly when there is also irregular fever and gradual loss of weight and strength. When exploration is out of the question it is worth trying treatment by penicillin and the sulphonamides, for there are few cases of actinomycosis which will not rapidly improve under this treatment.

Actinomycosis of the Kidney. Only about a dozen cases of primary actinomycosis of the kidney have been recorded. The symptoms are similar to those resulting from carbuncle of the kidney and it is unlikely that correct diagnosis will be made before exploration.

Nephrectomy has usually been performed for this condition. If it were possible to diagnose with certainty, then the condition should subside under appropriate drug treatment.

Pelvic Actinomycosis. In women the ovaries and Fallopian tubes have frequently been the site of actinomycosis, usually as an extension from an ileo-cæcal focus, but possibly (rarely) by infection extending up the cervical canal. The ovary appears to be a good medium for the growth of the actinomyces. Actinomycosis of the adnexa would scarcely be differentiated from septic infection of the tubes before operation. The surgeon would

have his suspicions aroused if the inflammatory process had infiltrated the pelvic wall and was of a gristly consistency. In spite of removal of the tubes and ovaries the suppurative process will almost certainly continue unless its true nature be recognized and treatment by appropriate drugs commenced. Pelvic actinomycosis differs from all other



(From "Actinomycosis" Zachary Cope, Oxford University Press)

Fig 201 Actinomycosis of liver resembling malignant growth (R.C S Museum)

pelvic inflammations in that it tends to penetrate through the sciatic notch into the buttock where an abscess forms and needs incision If not diagnosed earlier the true nature of the condition will then be recognised.

Treatment and Prognosis of Abdominal Actinomycosis

Formerly actinomycosis within the abdomen was of gloomy prognosis. Now that we have the antibiotics and the sulphonamides to help us the outlook is promising Provided these drugs be given in sufficient doses for long enough most cases should recover. The writer has found penicillin the most efficacious drug. Treatment should be continued for some weeks after all symptoms have disappeared.

CHAPTER XVI

RECTUM AND ANUS

SURGICAL ANATOMY

HENRY R. THOMPSON

THE rectum is continuous above with the sigmoid colon and ends below at the anal canal. Anatomically, it commences opposite the third sacral vertebra, its upper two-thirds being partly covered by peritoneum.

Pathological lesions in the segment of bowel from the true pelvic brim to the third sacral vertebra are in their behaviour similar to rectal lesions and it is convenient for descriptive surgery to recognize here a rectosigmoid zone where larger lesions may involve both rectum and colon.

The rectum is not, as its name implies, straight. It has antero-posterior and lateral curves.

The surgical anal canal commences at the point where the rectum passes through the musculo-fascial pelvic diaphragm of the levator ani muscle and terminates in a longitudinal antero-posterior slit. It is surrounded by the sphincter ani muscles.

The intestine, in common with other tubular structures in the body, has the basic structure of a lining, an areolar coat, and outer longitudinal and inner circular muscle coats of plain, unstriped, involuntary muscle.

At the termination of the intestine its components are modified; it is re-inforced with striped voluntary muscle and acquires a sensory nerve supply which brings the evacuation of the intestinal contents under the control of the voluntary nervous system.

The composite, complex muscular structure of the ano-rectal region is best understood by tracing the muscular components of the rectum to their termination.

Longitudinal Muscle Coat. As the longitudinal muscle coat passes down through the levator ani, fibres of the puborectalis portion of this muscle join it to form the conjoint longitudinal muscle. As this muscle passes downwards between the external sphincter and the circular muscle coat (internal sphincter) its fibres become fibro-elastic and pass inwards between the muscular fasciculi of the internal sphincter and outwards through the external sphincter muscle. The main body of fibres fan out and pass through the most superficial portion of the external sphincter to be inserted into the overlying perianal skin.

Fibres passing outwards between the superficial and deeper portions of the external sphincter muscle divide the ischio-rectal fossa into perianal and ischio-rectal spaces.

An anterior condensation of fibres is attached to the triangular ligament, to the urethra, and to the apex of the prostate and represents the recto-urethralis muscle.

Circular Muscle Coat. Below the levator ani, the circular muscle coat becomes thickened and forms the internal sphincter ani muscle. It varies in thickness and in length. Its lower border can be felt just within the anal canal and a distinct depression can be felt between it and the inner aspect of the subcutaneous external sphincter muscle—the ANAL INTERMUSCULAR DEPRESSION.

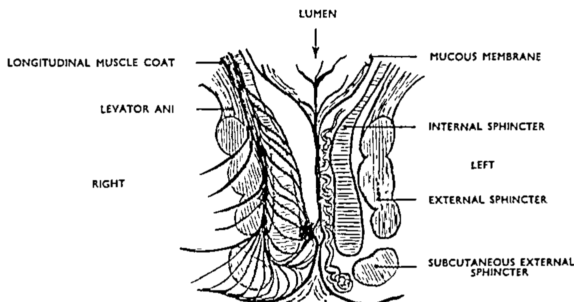


FIG 202. Coronal section through closed anal canal.
Right side showing terminations of conjoint longitudinal muscle. *Left side.* conjoint longitudinal muscle removed. Hæmorrhoidal varicosity in submucosa

LUMEN
LEVEL OF ANO-RECTAL RING
FIXED POINT OF ANAL MUCOSA
(a)

LUMEN
EXTERNAL SPHINCTER
INTERNAL SPHINCTER
SUB-CUTANEOUS EXTERNAL SPHINCTER
A
A'
VISIBLE INTER-HÆMORRHOIDAL DEPRESSION
(b)

FIG 203 Coronal section through partly opened anal canal

(a) Finger at palpable intermuscular depression Fixed point of anal mucosa to internal sphincter shown

(b) Showing formation of inter-hæmorrhoidal depression when a hæmorrhoid prolapses

(Longitudinal muscle omitted for clarity)

The External Sphincter Ani may be divided into a subcutaneous, a superficial and a deep portion. The subcutaneous external sphincter is an annular muscle band surrounding the anal orifice. Its fibres decussate anteriorly and posteriorly. Posteriorly the commissural fibres are occasionally replaced by muscle slips attached to the superficial external sphincter on either side.

The superficial external sphincter muscle is elliptical and attached to the coccyx behind and the perineal body in front. The deep external sphincter is annular and its upper margin can be defined anteriorly but posteriorly it blends with the puborectalis portion of the levator ani.

The three portions of the external sphincter muscle constitute a muscle tube which surrounds the anal canal and the corresponding muscle tube of the internal sphincter muscle. This relationship of external to internal sphincter as one tube within another must be emphasized. It is not, as has so often been depicted and described in the past, one muscle ring superimposed on the other.

Where the rectum passes through the levator ani muscle the ano-rectal ring is formed. It can be defined by palpation on contraction of the anal sphincters; it can be seen closing the anal canal over the end of a withdrawing proctoscope.

The ano-rectal ring is formed behind and at the sides by the sling of the puborectalis muscle and in front, where the puborectalis is deficient owing to its attachments to either side of the os pubis, by the deep portion of the external sphincter muscle.

The corresponding parts of the internal and longitudinal muscle coats of the rectum are included in this ring. It is destruction of this ring that will result in incontinence and preservation of a small slip is adequate for control of fæces.

The ano-rectal ring has in the past been confused with the internal sphincter muscle. In fistula operations the external and internal sphincter muscles can be divided provided the ano-rectal ring is left intact.

Lining of Anal Canal. The rectum is lined by a pink membrane of tall columnar mucus secreting cells. Being loosely attached to the underlying structures, it is thrown into folds in the upper part of the anal canal—the columns of Morgagni. In this region the columnar cells become cuboidal. The columns of Morgagni terminate in small crescentric valves and here the lining changes from cuboidal to transitional epithelium. The line formed by these valves has a scalloped edge. It has been referred to as the dentate, pectinate or valvular line. As mucous membrane changes to skin of anal canal at this point, it is also referred to as the muco-cutaneous junction. Below the valves the anal canal is lined by squamous epithelium, which is adherent to a condensation of longitudinal muscle fibres and submucosal tissue. It is smooth and thin and devoid of hair follicles or sweat glands. At the anal margin the anal canal skin merges into the true pigmented skin of anus with hair follicles and sweat glands.

Valves of Morgagni. These have practical significance: the pockets formed by the valves can become inflamed, giving rise to cryptitis. Anal intermuscular glands, small, vestigial, tubular, transitional celled structures, arise in the valvular zone and both conditions can be responsible for ano-rectal suppuration.

Valves of Houston. These are folds of mucous membrane, sometimes containing circular muscle fibres, usually three in number, a lower and upper fold on the left side and a middle fold on the right anterior aspect of the rectum. Small tumours may be obscured on their upper surfaces.

Blood Vessels of the Rectum and Anus

The rectum and anal canal derive their blood supply from three sources:

(1) The superior hæmorrhoidal artery, the continuation of the inferior mesenteric artery.

(2) The middle hæmorrhoidal artery, a branch of the anterior division of the internal iliac artery.

(3) The inferior hæmorrhoidal artery, a branch of the internal pudendal artery.

The venous return takes place by the corresponding veins; the inferior mesenteric vein drains into the splenic vein and thus into the portal circulation. The middle and inferior hæmorrhoidal veins drain into the internal iliac vein and thus into the systemic circulation. There is an anastomosis between the venules of these two circulations in the submucosa of the anal canal just below the valvular or dentate line.

Certain practical points must be noted about the arteries and veins of the rectum and anal canal.

Arteries. The distribution of the superior hæmorrhoidal artery determines the number and disposition of hæmorrhoids (Miles, 1939). The superior hæmorrhoidal artery at the level of the third sacral vertebra divides into a left and right branch. The right branch divides into anterior and posterior branches, the left branch has no main subdivision. It does, however, in common with the right posterior branch, have two small branches which arise just before the termination of the parent vessel. Because of this anatomical distribution the primary hæmorrhoids develop at right anterior, right posterior, and left lateral positions in the anal canal and secondary hæmorrhoids develop one on either side of the left lateral and right posterior hæmorrhoids.

The next practical point is the question of the blood supply to the rectal stump after excisions of the colon and upper rectum where continuity of the bowel is restored by an ileo- or colo-rectal anastomosis. When the rectum has been divided above the lateral ligaments and the inferior mesenteric or superior hæmorrhoidal arteries ligated, the middle hæmorrhoidal arteries are adequate blood supply to this length of rectal stump. When the rectum and lateral ligaments (containing the middle hæmorrhoidal arteries) have been divided and a short stump of rectum left above the levator ani muscle, the inferior hæmorrhoidal arteries are an adequate blood supply for this length of stump.

It is difficult to devascularize any rectal stump that a surgeon might wish to use for intestinal anastomosis performed in this region at the present time. The bogy of critical points and zones does not exist.

In elderly patients where the arterial tree has undergone severe athero-sclerosis, the question of the blood supply to the proximal and distal end of a colo-rectal anastomosis must be carefully considered in relation to the degree of arterial disease and there is no doubt that in these cases the same freedom of approach in ligating the blood vessels cannot be employed.

In combined excisions of the rectum the inferior mesenteric artery can always be divided below the left colic branch to leave an adequate blood supply to the colostomy and in the majority of cases it may be tied flush with the aorta, the left marginal branch of the middle colic artery also proving adequate blood supply to the colostomy. The middle hæmorrhoidal arteries can be identified and ligated during division of the lateral rectal ligaments as they pass from the pelvic wall to the rectum.

The inferior hæmorrhoidal arteries are divided during the perineal part of a combined

excision of the rectum as they pass across the ischio-rectal fossa and a large constant branch from the perineal branch of the internal pudendal artery is divided as it passes across the perineum to supply the anterior part of the sphincter ani muscles. This artery, because it is frequently divided during fistula operations, is known as "the artery of fistula."

Veins. Hæmorrhoids are varicosities of the superior hæmorrhoidal veins. When hæmorrhoids become infected or strangulated, or in any severe infection of the rectum, the possibility of infection spreading along the inferior hæmorrhoidal veins and causing a portal pyæmia is always present.

Rectal growths invade the tributaries of the inferior mesenteric vein and break off to pass through the portal circulation of the liver which explains why the liver is the most frequent site for rectal growth metastases. Because of this, the inferior mesenteric vein should be ligated before mobilizing a rectal growth. This important principle of cancer surgery, of shutting the venous escape route for cancer cells into the general circulation by ligating the venous drainage before the primary tumour is mobilized, is especially applicable to rectal growths. A certain amount of squeezing is unavoidable, especially in large advanced growths filling the pelvis where infiltration of growth down venules has probably already taken place.

As the superior hæmorrhoidal veins form a plexus under the anal mucosa to constitute the internal hæmorrhoidal plexus so the inferior hæmorrhoidal veins form a plexus under the skin of the anal canal to constitute the external hæmorrhoidal plexus.

Dilated veins of the external hæmorrhoidal plexus rupture and the extravassated blood clots to form a hard, rounded blue swelling known as an anal hæmatoma or external hæmorrhoid. This swelling may vary from the size of a small pea to an œdematous tumour involving the whole of one side of the anal margin. When extensive, an anal hæmatoma can be difficult to diagnose from a prolapsed thrombosed internal hæmorrhoid. With the latter a depression can always be observed between œdematous anal canal skin and the thrombosed internal hæmorrhoidal plexus. This depression is known as the INTER-HÆMORRHOIDAL DEPRESSION. It can also be seen in a simple prolapsing hæmorrhoid. It forms at the point where the anal canal skin is firmly fixed to the underlying submucosa and internal sphincter muscle in contrast to the loosely attached linings over the internal and external hæmorrhoidal plexuses.

Nerve Supply. The anal canal and its musculature are supplied from both the cerebro-spinal and autonomic nervous systems. Efferent impulses to the external sphincter muscle are transmitted down the internal pudic and perineal nerves deriving their fibres from the second, third and fourth sacral anterior nerve roots. Afferent impulses transmitting painful stimuli from the perianal skin and skin of the anal canal pass back along corresponding nerves.

The parasympathetic supply to the rectum arises from the second, third and fourth sacral nerve trunks, the nervi erigentes. Efferent impulses along these fibres cause the rectum to contract and the internal sphincter to relax. The sympathetic supply to the rectum comes from the inferior mesenteric plexus in front of the aorta and passes down the superior hæmorrhoidal vessels. It is also supplied by the pre-sacral nerve, from fibres of the second, third and fourth lumbar sympathetic ganglia. Efferent impulses along the sympathetic nerves relax the rectal wall and contract the internal sphincter. Afferent autonomic impulses convey sensations of distension. It is these impulses which

Blood Vessels of the Rectum and Anus

The rectum and anal canal derive their blood supply from three sources:

(1) The superior hæmorrhoidal artery, the continuation of the inferior mesenteric artery.

(2) The middle hæmorrhoidal artery, a branch of the anterior division of the internal iliac artery.

(3) The inferior hæmorrhoidal artery, a branch of the internal pudendal artery.

The venous return takes place by the corresponding veins; the inferior mesenteric vein drains into the splenic vein and thus into the portal circulation. The middle and inferior hæmorrhoidal veins drain into the internal iliac vein and thus into the systemic circulation. There is an anastomosis between the venules of these two circulations in the submucosa of the anal canal just below the valvular or dentate line.

Certain practical points must be noted about the arteries and veins of the rectum and anal canal.

Arteries. The distribution of the superior hæmorrhoidal artery determines the number and disposition of hæmorrhoids (Miles, 1939). The superior hæmorrhoidal artery at the level of the third sacral vertebra divides into a left and right branch. The right branch divides into anterior and posterior branches, the left branch has no main subdivision. It does, however, in common with the right posterior branch, have two small branches which arise just before the termination of the parent vessel. Because of this anatomical distribution the primary hæmorrhoids develop at right anterior, right posterior, and left lateral positions in the anal canal and secondary hæmorrhoids develop one on either side of the left lateral and right posterior hæmorrhoids.

The next practical point is the question of the blood supply to the rectal stump after excisions of the colon and upper rectum where continuity of the bowel is restored by an ileo- or colo-rectal anastomosis. When the rectum has been divided above the lateral ligaments and the inferior mesenteric or superior hæmorrhoidal arteries ligated, the middle hæmorrhoidal arteries are adequate blood supply to this length of rectal stump. *When the rectum and lateral ligaments (containing the middle hæmorrhoidal arteries)* have been divided and a short stump of rectum left above the levator ani muscle, the inferior hæmorrhoidal arteries are an adequate blood supply for this length of stump.

It is difficult to devascularize any rectal stump that a surgeon might wish to use for intestinal anastomosis performed in this region at the present time. The bogy of critical points and zones does not exist.

In elderly patients where the arterial tree has undergone severe athero-sclerosis, the question of the blood supply to the proximal and distal end of a colo-rectal anastomosis must be carefully considered in relation to the degree of arterial disease and there is no doubt that in these cases the same freedom of approach in ligating the blood vessels cannot be employed.

In combined excisions of the rectum the inferior mesenteric artery can always be divided below the left colic branch to leave an adequate blood supply to the colostomy and in the majority of cases it may be tied flush with the aorta, the left marginal branch of the middle colic artery also proving adequate blood supply to the colostomy. The middle hæmorrhoidal arteries can be identified and ligated during division of the lateral rectal ligaments as they pass from the pelvic wall to the rectum.

The inferior hæmorrhoidal arteries are divided during the perineal part of a combined

noting the state of the perianal skin, fistulous openings, tell-tale skin tags, hæmatomata, and prolapsed piles. The rectum is next palpated with the index finger covered by a glove or finger cot and a square lint shield. The finger should be lubricated with a water soluble lubricant (K Y jelly, Lubafax). Palpation of the rectum is not merely the insertion of the finger into the anal canal—the whole circumference of the rectum must be carefully palpated in its lower, middle, and upper thirds. Finally, the perineum must be pushed up, with the index finger *in situ*, and often the bowel can be examined as far as the sacral promontory.

While the right index finger is in the rectum, the left hand should palpate the lower

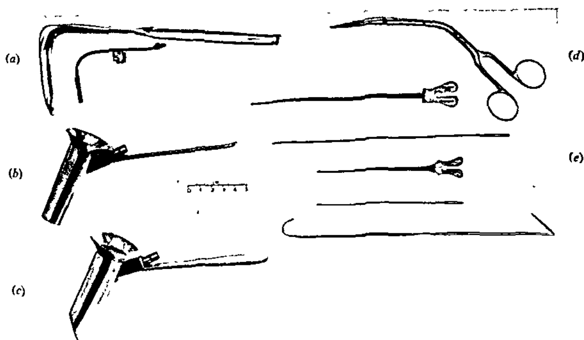


FIG 204

(*a*) Sims' pattern proctoscope with light attachment
(*b*) Tubular proctoscope.
(*c*) Graeme Anderson or Hirschman type proctoscope
(*d*) Caldwell-Luc forceps A suitable instrument for biopsy through a proctoscope
(*e*) Fistula probes

abdomen and the pelvis can thus be examined. Most anorectal lesions can be diagnosed by inspection and palpation.

Palpation is followed by proctoscopy and sigmoidoscopy. The most useful proctoscope is the tubular type with attached light. A modification where the end is cut obliquely (Graeme Anderson, Hirschman) allows the anal canal to be examined as the proctoscope is withdrawn. A pair of 8 in. dissecting forceps (Emmett's) for swabbing and an instrument for biopsy should be available (Fig. 204).

The full extent of hæmorrhoids can be better assessed if the patient is asked to strain down gently onto the proctoscope. The hæmorrhoids will then distend into the end of the instrument. If the hæmorrhoids are large they will prolapse when the proctoscope is withdrawn.

initiate the act of defæcation which is a highly complex synergism of these two nervous systems. Much remains to be understood about the working of both the external and internal sphincter muscles and there is no doubt, as suggested by Milligan, that the external sphincter has a double nerve supply which enables it to remain in tonic contraction which can be re-inforced at will. Another muscle in the body with a comparable action is the levator palpebræ superioris.

Lymphatic Drainage. This occurs mainly in an upwards direction through lymphatics and a chain of lymphatic glands along the inferior hæmorrhoidal vessels. At the origin of the inferior mesenteric artery, upward drainage is continued into the pre-aortic glands and finally glands at the root of the left side of the neck may become invaded by secondary growth from glands round the cisterna chyli and the cœliac axis.

Lymphatic drainage also occurs laterally along the lateral ligaments of the rectum and over the fascia on the levator ani muscle to involve glands of the pelvic wall. The anal canal drains outwards to the superficial group of inguinal glands and from thence to the deep inguinal glands and internal iliac glands.

Reference

Miles, W. E. (1939) *Rectal Surgery*, 1st Edition, Cassell, London, p. 122–128.

EXAMINATION OF A PATIENT WITH ANO-RECTAL DISEASE

It would be humbug to suggest that every patient presenting with rectal symptoms should be examined routinely from head to toe. A general examination of the patient must, however, always be borne in mind, especially at established rectal clinics where attention is closely focused on the rectum and anus. Constitutional diseases may present with ano-rectal signs and symptoms.

Before attending for ano-rectal examination patients should be reminded to evacuate the bowels.

The region is extremely sensitive, its exposure causes embarrassment to most patients who are also unable to see what the surgeon is doing. The surgeon must be gentle, sensitive to the patient's feelings, and be ready with a simple explanatory patter describing what he is going to do and what the patient may probably feel. The few extra minutes spent in this way secures a confident, co-operative, relaxed patient so essential for a complete and expeditious examination.

The three commonly used positions in which a patient with ano-rectal disease can be examined are (1) the left lateral or Sims' position, (2) the knee-chest position and (3) Hanes' or jack-knife position. The last position requires a special table; the first two can be used on a standard examining couch or the patient's bed. Every effort must be made to make the patient comfortable and there is no doubt most patients prefer the left lateral position, but it is not so convenient for the surgeon. In the knee-chest position the patient kneels on the couch with the legs apart, the left shoulder on the couch and the head turned to the left side. The back is then hollowed. When examined in this position collars and tight blouses should be loosened and spectacles should be removed and placed safely under the couch pillow.

The examination proceeds in an orderly fashion: careful inspection with a good light,

ANAL FISSURE

Definition. A fissure-in-ano is a split or tear in the skin of the anal canal.

Surgical Anatomy

It occurs commonly in the midline posteriorly, occasionally in the midline in front, and exceptionally to one or either side of the midline. Unless the anus is everted the fissure itself cannot be seen as it is placed in the lower ½–1 cm. of the anal canal and overlies the lower end of the internal sphincter muscle.

Longitudinal fibres can be seen in the floor of an acute fissure. These are the fibres of the corrugator cutis ani muscle derived from the conjoined longitudinal muscle.

As the fissure ulcerates deeper the white circular fibres of the internal sphincter muscle are exposed and occasionally both sets of muscle fibres can be seen at the same time.

Pathology. The passage of a large hard stool through the anus with overstretching and splitting of the skin of the anal canal would appear to be the commonest cause. It can occur, however, during attacks of diarrhoea. Young women in the puerperium are prone to develop anterior fissures, presumably due to stretching and weakening of the perineum during labour.

Prolapse of a large fibroid polyp may also tear the anal skin, the fissure so produced being a little lateral to the midline.

The anal skin varies in texture in individuals and can be so fragile that gentle traction to either side of the anal margin may cause a split.

As similar sized lesions on exposed surfaces heal rapidly there must be local factors to explain why an anal fissure so frequently fails to heal. It is due to the fact that reflex spasm of the sphincter keeps the anal canal tightly shut and the inflammatory exudate cannot drain away but remains in contact with the fissure. As a result the fissure edges become undermined, especially at its base where a subcutaneous abscess may form. If the abscess bursts and discharges a fistula results, its track frequently becoming epithelialized.

The so-called "sentinel pile" or skin tag can be seen in acute or chronic fissures and in the first place it is probably formed by skin from the injured anal canal; with secondary inflammatory changes this tag becomes œdematous and swollen and contributes to the failure of healing.

A small "pseudo polyp" of anal mucosa may be present at the apex of the fissure.

When a fissure becomes chronic secondary changes take place in the internal sphincter muscle. Chronic inflammation terminates in fibrosis and the internal sphincter lying in the floor of the fissure is involved in this change. The patient, moreover, to avoid discomfort maintains his stools in a semifluid state and the dilating effect of a formed stool is lost.

Signs and Symptoms. Severe, cutting pain during and after defæcation is the main symptom. This pain may last for an hour and can be so distressing as to make a strong man faint. Slight bleeding and discharge frequently occur and the patient may be conscious of a swelling at the anus. The discharge produces secondary changes in the perianal skin and is a cause of pruritus ani.

The presence of a sentinel pile with strong sphincter spasm is an external sign. Before examining the anus digitally or with instruments a surface anæsthetic should be applied

The simplest instruments are invariably the best, and the Lloyd-Davies type of proximally lighted sigmoidoscope is no exception. It is easy to use, adaptable, and stands up to hard wear and tear. Barrels of different length and calibre can be readily interchanged and a magnifying lens can be attached for detailed study of the bowel mucosa. Again, with the sigmoidoscope, a pair of swab-holding and biopsy forceps is required. *Patterson's forceps combine these functions adequately (Fig. 205).*

Routine preparation of the bowel for sigmoidoscopy is a mistake. The presence of blood, pus, and mucus in the lumen of the bowel are valuable clues to diagnosis, and preparation may remove such evidence. The mucosa of the rectum and colon become

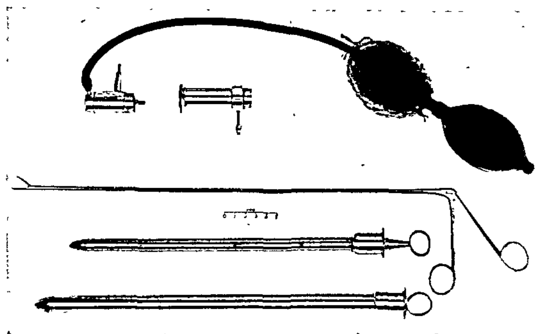


FIG. 205 Lloyd-Davies sigmoidoscope with magnifying lens attachment and Patterson combined swab-holding and biopsy forceps

hyperæmic after enemata and this colour change may be mistaken for an early proctitis or colitis.

Two glycerine suppositories inserted in the rectum and a wait of fifteen minutes will usually lead to a successful evacuation in patients who attend for examination with the rectum full of fæces. Only after repeated failed examinations should the bowel be prepared by washouts.

Patients with acute painful lesions are generally tensed, with the sphincter ani in spasm. These patients can be successfully and painlessly examined if a local anæsthetic jelly (Xylocaine 2 per cent) or ointment (Decicain 10 per cent) is gently inserted into the anal canal on a throat swab and followed by a similarly lubricated finger. The painful lesion is rendered anæsthetic, the sphincter relaxes, and a full examination can generally be made. (*See* Chapter XII, Neonatal Intestinal Obstruction.)

Conservative measures sometimes fail to heal an acute fissure-in-ano and relapse is not uncommon.

Operative Treatment

A patient with an anal fissure asks for relief from the dread, fear, and pain of daily defæcation. Treatment, therefore, should be simple and, if possible, immediately available without the wait for a hospital bed.

The lesion is a minor lesion and patients often procrastinate if a period of "in-patient" treatment is recommended. The fissure becomes chronic and only the advent of complications persuades the patient to come into hospital.

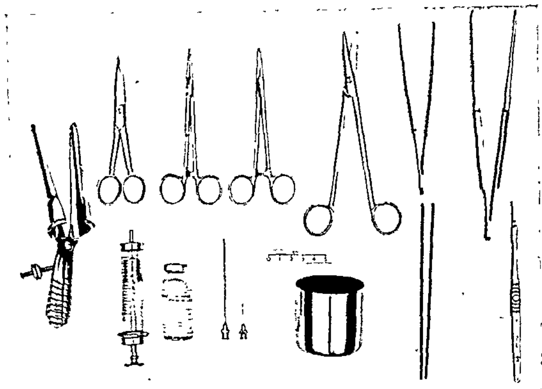


FIG 207 Instruments for sphincterotomy, Ricord bivalve speculum

Treatment which will relieve symptoms quickly is, however, available and results in a permanent cure.

As emphasised by Eisenhammer (1951) the key anatomical structure in the treatment of anal fissure is the internal sphincter ani muscle. Division of this muscle results in painless healing of the fissure.

The internal sphincter can be divided under local anæsthesia in the out-patient department at the time of consultation and for the majority no in-patient treatment is required.

Domestic factors in individual cases may necessitate admission for 24–48 hours. If the patient is nervous and apprehensive, has a long return journey and no help at home, it is better to admit him for a short period.

to the fissure. The ulcer can then be inspected; the presence of undermined edges, polypi, fistulæ, and hæmorrhoids is noted. They play a part in determining treatment.

A tubular proctoscope of the Graeme Anderson or Hirschman pattern with an oblique end gives a clear view of the anal canal and fissure area.

Differential Diagnosis is from other chronic ulcers of the anal canal. Traumatic, tuberculous, syphilitic ulcers and squamous celled carcinoma have similar symptomology. These ulcers, besides having their own characteristic features, are not as a rule midline ulcers and sphincter spasm is not so marked.



FIG 206 St Mark's Hospital conical dilator

Conservative Treatment. An acute fissure will heal aided by local applications and injections of anæsthetic solutions into the tissues round the anal canal.

People who develop fissures are often tense, nervous patients and medication with Phenobarbitone gr. ½ twice a day during treatment is of great help. There is a natural tendency for patients to purge themselves or confine the bowels which should be regulated to a daily soft, formed stool.

At the time of examination a drop of pure phenol, ichthyol or silver nitrate should be applied to the ulcer. The patient is instructed to introduce Decicain ointment 3 per cent into the anus. This may be done conveniently on the end of a St. Mark's Hospital conical dilator. This dilator is passed twice a day for 14 days. After this time the lubricant is changed to zinc and castor oil (B.P.) or scarlet red ointment. If anæsthetic ointments are used for longer than 2–3 weeks a sensitization dermatitis may develop in the perianal skin.

Injections of oily anæsthetic solutions such as Proctocaine (Allen and Hanbury's) or Nupercaine in oil (Ciba Co.) give immediate relief of pain and the effect may last for 2–3 weeks. The injections are given into the tissues around the anus, through a puncture in the skin one inch behind the posterior anal margin.

The patient lies in the right lateral position, the skin is shaved and carefully prepared with Cetavlon, spirit, and iodine. Five ml. of solution are introduced into either side, keeping the point of the needle moving while the injection is being made to avoid pooling the solution.

Surgical asepsis must be strict; introduction of sepsis can lead to the formation of troublesome sub-acute abscesses and the injection must not be made in the presence of a fissure-abscess or fistula.

Following these injections patients should be warned that there may be a temporary loss of control of defæcation and passing flatus. The degree of incontinence may be very slight and is only rarely complete.

After the bowels are open the patient has a bath and the dressing consisting of a gauze swab moistened with 2½ per cent Milton solution is tucked into the anal canal and maintained in place with a "T" bandage.

On the fifth post-operative day a St. Mark's Hospital dilator is passed and thereafter twice a day until the fissure has healed. During the healing period the patient should take Phenobarbitone gr. ½ three times a day.

It is advisable to take a week off work but many patients who run a "one man business" are back in 3 days.

Healing can be expected in 14–21 days but during the healing period the wound is relatively painless.

Unless a long enough drainage incision is made posteriorly an area of exuberant granulations will develop inside the anal canal which, although painless, will give rise to bleeding, discharge, and perianal irritation. These granulations can become very indolent but will heal with frequent applications of solid silver nitrate.

Precautions. If the fissure is situated anteriorly the internal sphincter should be divided in the left posterior quadrant; anteriorly situated wounds of the anal canal do not heal well.

If hæmorrhoids are present to any degree, on no account should the operation be done as an out-patient. The patient should be admitted and the hæmorrhoids and fissure treated at the same time.

Reference

Eisenhammer, *South African Med. Jour*, 1951, 25, 486.

HÆMORRHOIDS

Hæmorrhoids or piles (from the Latin pila, a ball) are names unfortunately used universally to account for any symptom referable to the anus and rectum.

Hæmorrhoids are varicosities of the tributaries of the internal hæmorrhoidal veins and are situated under the mucosa of the upper half of the anal canal. They communicate with the veins of the external hæmorrhoidal plexus situated under the skin of the anal canal.

Traditionally, hæmorrhoids are divided into external hæmorrhoids and internal hæmorrhoids. By "external hæmorrhoid" is understood a swelling outside the anus and thus the term has been applied to pathological conditions other than hæmorrhoids. These are:

(1) ANAL HÆMATOMATA. Congestion due to constipation can result in dilated veins of the external hæmorrhoidal plexus rupturing and forming a hæmatoma under the skin of the anal canal. The patient is aware of a tense tender swelling at the anal margin. Untreated, the hæmatoma gradually subsides, becomes symptomless in 10–14 days, and disappears in 14–21 days according to its size. It may leave a skin tag. Hæmatomata are frequently multiple. In women they occur particularly at period time.

If seen in the first 48 hours the anal skin over the hæmatoma is infiltrated with local anæsthetic and the clot gently expressed. Washing the perineum night and morning with a thick soap lather is all the post-operative treatment required. By careful history-taking an effort should be made to correct the conditions leading up to this small vascular episode; if this is not done, further hæmatomata may develop.

Internal Sphincterotomy

Preparation. Only in the presence of a loaded rectum is there any necessity to prepare the bowels. Then the insertion of 2 glycerine suppositories into the rectum will be followed in 15–20 minutes by a satisfactory evacuation.

The perianal skin is shaved and cleaned with Cetavlon, spirit, and iodine. A small quantity of 10 per cent Decicain (Bayer) ointment is inserted into the anal canal on a throat swab to produce a surface anæsthesia.

With the patient in the right lateral position a weal is raised one inch posterior to the anal margin with 2 per cent Xylocaine (Duncan and Flockhart) with adrenaline. The perianal tissues are then infiltrated on each side with 10 ml. of 2 per cent Xylocaine. Five minutes should be allowed for the anæsthetic to take maximum effect.

The patient then turns into the left lateral position. A well-lubricated Ricord bivalve speculum is gently inserted into the anal canal and slowly opened to expose the fissure. The fingers of the left hand pull back the skin posteriorly to evert the anal canal and further expose the fissure.

With a small scalpel blade an incision is made through the fissure (if situated posteriorly), the longitudinal fibres of the corrugator muscle (if present) are divided, exposing the white circular fibres of the internal sphincter; this muscle is now divided to just above the pectinate or dentate line. This exposes a smooth tissue plane which is the main part of the conjoined longitudinal muscle.

The incision is carried outwards into the perianal skin posteriorly and because of the eversion of the anal canal this may not be made sufficiently long. It is essential, for drainage, to have one inch of incision showing when the speculum has been removed and the anus is closed.

The commissural fibres of the subcutaneous external sphincter may be exposed and, if well developed, a few fibres may require dividing at the anal margin to "flatten" the wound.

Anatomical details can be clearly defined and better demonstrated if the wound is gently swabbed with adrenaline during the operation.

If skin tags and pseudo polyps are present they should be removed with minimal sacrifice of tissue. Small fistulæ, often with epithelialized tracks, must not be overlooked but laid open.

Only exceptionally is there any bleeding which needs to be stopped by forcep pressure.

Before the speculum is removed a small piece of Oxycel gauze is placed on the wound and a corner of a gauze swab moistened in 2½ per cent Milton solution is inserted into the anal canal

The patient must wait half an hour, and the dressing be then carefully examined, before returning home.

Post-operative Care

A patient operated on in the morning returns home and goes to bed, taking two Codeine tablets. The same night, before retiring to sleep, Pethidine 100 mgm. is taken and in the early morning a further two Codeine tablets may be required.

The next day the patient may be ambulatory and should take half an ounce of liquid paraffin night and morning with the object of having the bowels open on the following day.

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If the patient does not seek early advice symptoms are probably abating and surgery will only temporarily aggravate the patient's discomfort. Compresses of Lotio Plumbi Evaporans (B.P.) will hasten resolution and should be applied at least three times a day.

(2) The *skin tag* of anal fissure is often referred to as a sentinel pile or external hæmorrhoid.

(3) FIBROUS POLYPI, the result of long standing hæmorrhoids, are labelled external hæmorrhoids.

(4) SKIN TAGS. These are seen in conditions unassociated with hæmorrhoids as the legacies of hæmatomata, pruritus ani, and ano-rectal infections.

Redundant skin may form over the varicose external hæmorrhoidal plexus during the development of true internal hæmorrhoids and correspond to the anatomical location of the three primary piles. They can be felt by the patient and seen on examination. When hæmorrhoids prolapse they are correctly described as intero-external hæmorrhoids.

An anal hæmatoma may be the first sign of a carcinoma developing higher up in the ano-rectum. The diagnosis of a hæmatoma by inspection should not cut short a routine examination. An early squamous carcinoma at the anal margin is often incorrectly diagnosed as an "external pile."

Ætiology. Internal hæmorrhoids are rarely seen before the age of 20 years. It is therefore reasonable to suppose that they are acquired and that a factor or factors yet unknown must be responsible for their development over a period of time. The fact that they occur in families by no means implies a hereditary factor. The condition is common and bad habits are passed on from parents to offspring.

There is no occupation in which hæmorrhoids give rise to a claim for compensation. They frequently develop during pregnancy and are an unpleasant complication of childbirth; it is difficult to imagine any comparable factors in the male which would lead to their development by similar obvious means. Conditions where the pressure in the portal venous system is known to be raised are relatively uncommon and hæmorrhoids (except œsophageal) are by no means clinical features of these diseases. The seasonal incidence at Christmas and the New Year suggests that dietary excesses and irregular habits play a part in development.

The fact remains that the time honoured ætiological factors are largely supposition.

Theories are necessary as a basis for treatment and with early hæmorrhoids the question of arresting their development arises. A patient with early hæmorrhoids should be advised to keep his rectum empty (a natural state). This implies a bowel habit regulated not by domestic considerations but by the physiological reflexes of the gastro-intestinal tract. It also implies regular meals of balanced quality and moderate quantity.

After breakfast evacuation of the bowels should be a short business-like affair; not a period of prolonged intermittent straining with a pipe and *The Times*, nor the hurried, explosive evacuation of liquid fæces resulting from unwise purgation.

Simple advice which can be naturally applied is seldom popular or heeded and it is possible that neglect of the obvious accounts for the multitude of preparations and large quantities of aperients used today and the high incidence of hæmorrhoids and other anal lesions.

Symptoms. The first symptom is bleeding during defæcation, blood spurting from the compressed congested hæmorrhoid, and spotting the porcelain of the lavatory pan (first degree hæmorrhoids). Later the piles start to prolapse during defæcation, returning

spontaneously when the levator ani contracts at the end of defæcation. This is effected by virtue of its connection with the longitudinal muscle coat of the rectum and as the name levator ani suggests, it draws the anus in and the prolapsing piles back into place (second degree hæmorrhoids).

Finally, the hæmorrhoids prolapse but the longitudinal muscle and its attachments are so stretched that spontaneous return does not occur and moreover they prolapse apart from defæcation, during lifting efforts, coughing and sneezing, and even with mild physical exercise such as walking (third degree hæmorrhoids). Associated with third degree hæmorrhoids are a mucous discharge and pruritus ani. Pain is not a symptom of hæmorrhoids in an uncomplicated state.

Surgical Anatomy and Pathology. A hæmorrhoid consists of a group of varicosities of the internal hæmorrhoidal plexus supplied by a branch of the superior hæmorrhoidal artery and covered by the mucosa of the anal canal. The internal hæmorrhoidal plexus drains up the superior hæmorrhoidal vein into the inferior mesenteric vein to terminate in the splenic vein. It is a long vein and without valves.

The superior hæmorrhoidal artery is a direct continuation of the inferior mesenteric. It divides at the level of the third sacral vertebra into right and left branches. The right branch further subdivides into anterior and posterior branches and the right posterior branch gives off two further branches. The left does not subdivide but gives off two small branches comparable to the right posterior branch.

This arterial distribution accounts for the number and location of the hæmorrhoids. There are three primary piles:

(1) Right anterior.
(2) Right posterior.
(3) Left lateral.

There are four secondary hæmorrhoids, two associated with the left lateral hæmorrhoid and two with the right posterior hæmorrhoid. They are:

(1) Left anterior.
(2) Left posterior.
(3) Right lateral.
(4) Posterior in the midline posteriorly.

In common with other arteries the superior hæmorrhoidal may vary in the distribution of its branches with a corresponding variation of the anatomical arrangement of the primary and secondary hæmorrhoids.

The anal mucosa of long standing prolapsed hæmorrhoids may undergo a squamous metaplasia. Recurrent attacks of thrombosis followed by fibrosis of the clot results finally in the formation of a fibrous polyp.

Complications

(1) Thrombosis can occur in early piles and sometimes there is difficulty in diagnosing between an anal hæmatoma and a thrombosed pile.

(2) The combination of prolapse and sphincter spasm may result in gangrene of prolapsed piles.

(3) Ulceration and gangrene give rise to sepsis and anorectal abscesses result from suppuration in the anorectal lymphatic glands.

(4) Portal pyæmia may develop as a sequel to gangrenous piles.

(5) Prolonged and repeated bleeding results in severe degrees of anæmia. So insidious is its onset that the patient is often unaware of the secondary effects of the hæmorrhoids until dyspnœa, swelling of the legs, and a sore tongue develop.

Treatment

(*a*) MEDICAL. For early first degree hæmorrhoids education in bowel habits and dietetics may save a patient from the later stages and wean him from aperients.

(*b*) INJECTION TREATMENT. The object is to sclerose and obliterate the varicose veins

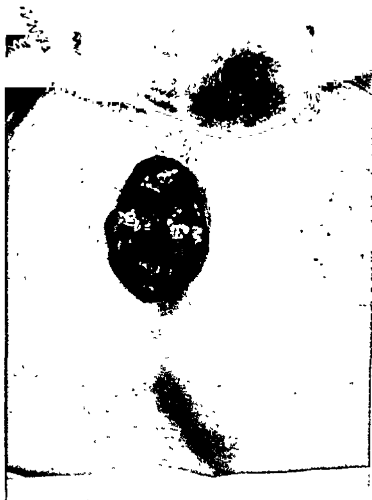


FIG 208 The three primary hæmorrhoids

and to draw up and fix down the loosened mucosa to the muscle coats of the rectal wall. The most satisfactory and safe solution to use is Albright's solution of 5 per cent phenol in almond oil with two grains of menthol to the ounce.

The injections are given without anæsthetic with the patient in the left lateral or knee chest position.

A lighted Kelly's tubular proctoscope is passed into the rectum and withdrawn until the anorectal ring of muscle is seen closing over the proctoscope. This is the site of the pile pedicle and the initial injections are given into the pedicles of the three primary

hæmorrhoids just above the anorectal ring. Three to five ml. of solution are injected into the submucosa which will distend into a bluish cyst with capillaries running over the surface.

Subsequently the hæmorrhoid bases are injected and, lastly, the interhæmorrhoidal areas. On an average, therefore, nine areas require to be injected.

Depending on the size of the hæmorrhoids, one or multiple injections may be given. If only one site is injected, other areas may be injected at weekly intervals. If three areas are injected it is advisable to wait for 2–3 weeks before a further injection is made.

During the injection, which should be painless, the patient may have an uncomfortable feeling of distension. He must subsequently be warned that an hour after injection a dull ache may be felt and a desire to defæcate must be resisted until the morning after injection. A hot bath and two Veganin tablets suffice to ease the ache.

Complications

(1) With large injections (15 ml.) the patient may experience momentary dizziness.

(2) Injection ulcers result from injecting into the mucosa. The tissue turns white and if this is seen to occur the injection must stop immediately. Constitutional symptoms of malaise and headache, usually described by the patient as a 48 hour attack of influenza, are evidence of a misplaced injection. The ulcer, however, heals in 2–3 weeks and the additional fibrosis gives an excellent result.

(3) Where second courses are necessary there is a tendency for a re-injected hæmorrhoid to bleed profusely from the needle puncture. It can be seen directly the needle is withdrawn and must be stopped before the patient leaves the examining couch.

(4) Genito-urinary complications. Injections into the right anterior hæmorrhoid in the male may be made too deeply (usually due to a blunt needle) into the prostate gland, urethra, or seminal vesicle. The patient may pass blood or oil down the urethra and a prostatic abscess or urethral stricture, attended with all their sequelæ, may develop. Dickson Wright (1950) has described these complications

(5) Accidental Intravenous Injections. Several cases have been reported of acute upper abdominal pain followed by enlargement of the liver and transient jaundice and there seems to be no doubt that this is secondary to the accidental injection of the solution into the portal system via the hæmorrhoidal veins. It should be borne in mind as a differential diagnosis if patients complain of acute abdominal symptoms following hæmorrhoid injections. Occasionally patients complain of being able to taste and smell carbolic after injections. The mode of product of this system is obscure.

(*c*) Operative Treatment should be reserved for third degree hæmorrhoids in patients over 40 years of age. The aim of operative treatment is to excise the external hæmorrhoidal plexus and ligate the internal hæmorrhoidal plexus at the pile pedicle. This will result in a permanent cure without the formation of skin tags which result from post-operative thrombosis of the external hæmorrhoidal plexus.

Pre-operative treatment aims at presenting the patient for operation with an empty colon and rectum and can be achieved without fuss by enemata and aperients according to the patient's habits

The operation described by Milligan and Morgan (1937) has stood the test of time.

Local anæsthesia with Xylocaine 2 per cent combined with thiopentone or low spinal

anæsthesia are ideal, the disadvantage of the latter being a larger percentage of urinary complications and a 5 per cent incidence of spinal headache.

The pedicles of the primary hæmorrhoids are grasped with hæmostats and drawn down; the skin over the corresponding external hæmorrhoid is tensed by a second forcep and two incisions made through the skin of anus and on either side of the anal canal. The fibres of the corrugator cutis ani muscle are divided, exposing the lower border of the internal sphincter muscle and the fibres of the longitudinal muscle passing through the internal sphincter are identified. The anal mucosa is divided and the mucosa at the pile pedicle is tied to the longitudinal muscle fibres with a strangulating ligature of No. 16 plaited silk. This fixes the pile pedicle at the lower border of the internal sphincter and reduces the raw area of the anal canal left to granulate. When fibrosis and contracture of the internal sphincter has occurred the muscle should be divided in the left lateral wound.

At completion of the operation there are three wounds separated by three bars of anal skin and mucosa in continuity with rectal mucosa. The interhæmorrhoidal bars of skin are a safeguard against anal stricture which in the past was a not uncommon complication.

Post-operative Treatment. The bowels are opened on the third post-operative morning with aperients and a gruel and olive oil enema. Thereafter the wounds are irrigated night and morning with 2½ per cent Milton solution. On the sixth post-operative day a finger is passed and subsequently a St. Mark's dilator at the time of each dressing. The dressing consists of a corner of gauze moistened with 2½ per cent Milton, gently inserted into the anal canal along an interhæmorrhoidal bar. The patient takes a bath before each dressing

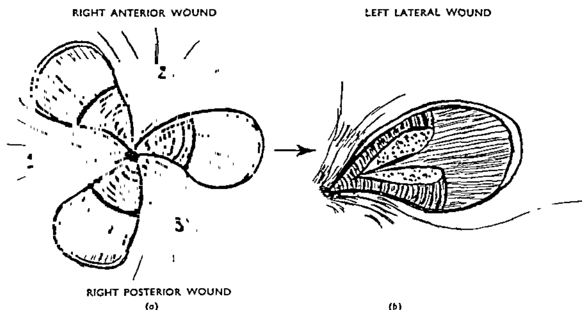


FIG. 209 (*a*) Anus at completion of operation for hæmorrhoids, showing (1, 2, 3) the three interhæmorrhoidal bars and intervening wounds with internal sphincter muscle exposed
(*b*) Left lateral wound showing division of internal sphincter muscle.

The stay in hospital is 10–14 days and the wounds are usually healed in three weeks when use of the dilator may be discontinued.

Complications

(1) *Reactionary hæmorrhage*, after the patient returns to the ward, is due to a slipped ligature.

(2) *Secondary hæmorrhage* (6–10 days). The rectum should be packed with vaseline gauze wound round a rubber tube and inserted through a proctoscope.

(3) *Skin tags*, which in the past were almost inevitable, should not occur. They are the result of thrombosis of the external plexus and secondary œdema of the overlying skin. Although of little clinical significance, they are of great discomfort and irritation to the patient who rightly expects a smooth anus and regards them as evidence of a badly performed and incomplete operation. If small, resolution will rapidly occur; a large skin tag, however, is better excised under local anæsthesia between the seventh and tenth post-operative day.

(4) *Abscess and fistula formation* are occasional sequelæ to hæmorrhoidectomy. They may occur round ligature knots and for this reason ligature ends must be left long or hæmostasis secured by diathermy.

(5) *Stricture at the anus*, in the past a frequent and troublesome complication, can be eliminated by leaving adequate interhæmorrhoidal bars and by careful supervision of after treatment.

Results of Treatment

Injection therapy is suitable for first and second degree hæmorrhoids and can give satisfactory long term cures. A further course of injections may be needed in 3–5 years' time. Injections can also give gratifying results in third degree hæmorrhoids.

Hæmorrhoidectomy has a bad reputation as a painful unsatisfactory operation, with the result that many people suffer piles and their effects for many years. They live in the shadow of a daily dread of defæcation, inseparable from aperients, and in a state of chronic ill health. By gentle handling of tissues an operation on the sensitive anus can be made tolerable and permanent relief from chronic discomfort and ill health achieved.

References

Dickson Wright, A (1950) *Proc. Roy Soc. Med* 43, 263.
Milligan, E. T. C.
Morgan, C. N. *et al*
Lancet (1937) 2, 1, 119.

INJURIES TO RECTUM

FOREIGN BODIES

Civilian Injuries

Injuries to the rectum in civilian life can occur from ingested foreign bodies or from penetration of the rectum from below.

It is remarkable how rabbit bones, poultry bones, fish hooks, and pins can negotiate the length of the human intestine without incident until reaching the rectum. Here they occasionally pierce the rectal wall and cause an ano-rectal abscess. The patient may

experience a sharp stabbing pain at the moment of defæcation, to be followed in a day or two by malaise and the throbbing pain of localized pus. Rabbit bones have become impacted across the rectal lumen to form a granuloma almost identical with a carcinoma.

Penetrating injuries of the rectum have occurred during sigmoidoscopy and during endoscopic removal of polypi from the anterior wall of the upper third with the diathermy snare. Too much traction is applied to the polyp which is snared too near its base and a circular hole in the rectum results. After removing a polyp with a diathermy snare the

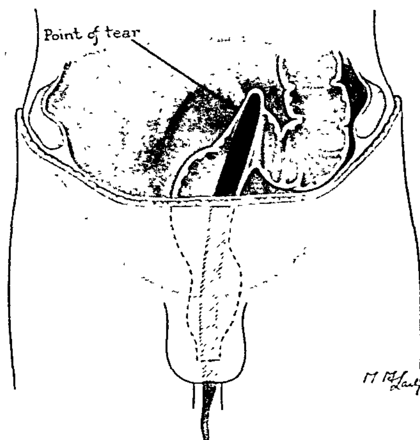


FIG 210. Diagram of a home-made bougie injuring lower colon. This bougie had been passed daily for two years with satisfactory results before injury occurred

area of removal must be inspected to exclude this injury and the occurrence of hæmorrhage.

Injuries from enema nozzles are not infrequent. The rectum may be perforated and a pelvic cellulitis result, or an interstitial injury result in enema fluid stripping up the component walls and causing gangrene of the rectum.

Foreign bodies are sometimes introduced into the rectum by the patient himself: home made bougies to assist defæcation, and a variety of objects from screwdriver handles to inverted tumblers used to relieve anal irritation and replace prolapsed piles. It is also a habit of anal neurotics.

These practices may continue for many years without an incident but unexpectedly the bowel may be perforated or the foreign body inserted too far.

Many mistakes are made, especially in the removal of glass foreign bodies, chiefly because an atmosphere of desperate urgency develops and insufficient thought and care are given to working out details for the removal and the risks involved. It is the first move taken in these cases which will determine between an uncomplicated removal or a surgical disaster and multiple operations.

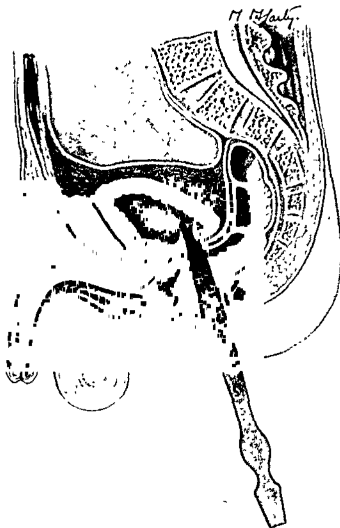


FIG 211 Impalement injury of rectum and bladder by a broken chair leg in a boy aged 9 years Note sub-peritoneal hæmorrhage

The rectum may be injured during prostatectomy, especially when diathermy is used for hæmostasis in the prostate bed and a recto-urethral fistula develops.

Third degree tears involving the rectal sphincter occur in difficult childbirth Fortunately they are amenable to treatment and incontinence need never be permanent.

Children who climb fences and railings are exposed to the risk of impalement injuries Spikes and palings penetrate through the rectum and frequently perforate the urinary bladder Extensive hæmorrhage into the pelvic cellular tissue and under the peritoneum of the pelvic floor and lower abdomen give rise to rigidity suggesting an intraperitoneal lesion.

In all injuries of the rectum where sphincter damage is extensive and continence imperilled a colostomy should be performed. If the bladder is injured it must be drained by suprapubic cystostomy.

Battle Wounds of the Rectum are caused by gun-shot wounds and shell wounds of the buttocks. They may be extra- or intra-peritoneal. Large metal fragments can cause extensive damage to rectum and bladder, to the lumbo-sacral plexus, with destruction and splintering of the sacrum and pelvic bones.

Treatment consists of removal of foreign bodies, arresting hæmorrhage, provision for drainage of pelvic cellular tissue, defunctioning of the rectum by colostomy and the bladder, if injured, by suprapubic cystostomy. If the lesion is intraperitoneal the small intestine must be searched for perforations and damage to other viscera.

FISTULA-IN-ANO

A fistula-in-ano is an abnormal fibrous track lined by granulation tissue leading from the anal canal to the surface.

Ætiology. A fistula results from ano-rectal infection and suppuration and in the majority of cases is of simple pyogenic origin. The infection may arise in one of the common ano-rectal lesions, an infected anal hæmatoma, a fissure, a thrombosed ulcerated hæmorrhoid. An abscess of the ischio-rectal fossa sometimes arises in circumstances which suggest a blood stream infection.

In some cases, however, there is no history of an acute abscess and infection from the anal crypts and anal intermuscular glands undoubtedly is responsible for "quiet" fistulæ. The majority of fistulæ heal quickly with adequate treatment; in a residue healing is not achieved or is delayed. While incorrect treatment is largely responsible for this failure of healing, it may be secondary to certain specific pathological conditions which are listed below:

(1) Presence of an unsuspected foreign body.
(2) Anal intermuscular glands incompletely removed.
(3) Tuberculous infection.
(4) Apocrine gland infection.
(5) Endometriosis
(6) Ulcerative colitis
(7) Crohn's disease or regional ileitis.
(8) Actinomycosis.
(9) Osteomyelitis of pelvis with sequestrum formation.
(10) Lymphogranuloma.
(11) Appendix abscess.
(12) Carcinomatous change.

In apocrine gland infection the whole area of infected skin must be excised and the wound skin grafted.

Endometriosis occurs in women in the perineal region in front of the anus. Exacerbation of symptoms at period time is the obvious clue to diagnosis. Clumps of uterine endometrium are found on microscopic examination of the excised tissue. It is difficult to achieve a radical dissection and the question of artificial menopause must be considered.

A fistula may result from intra-abdominal lesions such as Crohn's disease, intestinal tuberculosis, ileocæcal actinomycosis, chronic appendix abscess, tubo-ovarian abscess,

and diverticulitis. Abscess formation occurs in the supra levator space, spreads through the levator ani into the ischio-rectal fossa and thence to the skin. The resulting fistula, a pelvi-rectal fistula, is fortunately uncommon. Tuberculous and actinomycotic infection can occur also as a local lesion.

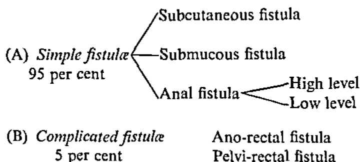
Crohn's disease has a bad reputation of presenting as a fistula without any of the recognized symptoms of enteritis. It should always be suspected when a well treated fistula is tardy or fails to heal. Occasional bouts of colic and diarrhœa are of diagnostic significance and it is unwise to wait for positive radiological findings which are notoriously unreliable. Laparotomy should be made on clinical grounds.

There are many ways in which a carcinoma and fistula are associated. Mucoid carcinoma has a tendency to burrow and form an atypical growth with multiple fistulæ. Growths in the recto-sigmoid may implant on the granulations of a chronic fistula and there is no doubt that very long standing fistulæ can undergo malignant degeneration.

Anatomy of Fistula

A fistula consists of a main track, one or more internal openings, one or more external openings, and subsidiary tracks. Infection spreads along the fibres of the longitudinal muscle as they pass through the external and internal sphincter muscles from the anal submucosa to their attachments to the perianal skin.

Fistulæ may be classified into:



The classification is based on the relationship of the main track to the anal musculature. In simple fistulæ the main track lies below the ano-rectal ring and radially in relation to the anal canal. In complicated fistulæ the main track lies parallel to the anal canal and is partly above the ano-rectal ring.

Simple Fistulæ. The tracks of subcutaneous and submucous fistulæ do not traverse any part of the sphincter ani muscle. Low anal fistulæ have their internal opening at the anal intermuscular depression, and traverse a part or the whole of the subcutaneous external sphincter. High anal fistulæ may have one or more internal openings between the anal intermuscular depression and the ano-rectal ring. These openings are always below the ano-rectal ring and traverse a variable amount of both internal and external sphincter muscles.

Subcutaneous, submucous, and low anal fistulæ account for 80 per cent of fistulæ. High anal fistulæ account for 15 per cent.

Complicated fistulæ comprise 5 per cent of fistulæ.

(1) Ano-rectal fistula is the fistula resulting from infection of the ischio-rectal fossæ, the so-called ischio-rectal or horseshoe fistula. The main track passes upwards to the

apex of the ischio-rectal fossa and can be felt above the ano-rectal ring. The internal opening (which may be multiple) is always below the ano-rectal ring.

(2) Pelvi-rectal fistula—the internal opening is above the ano-rectal ring and results from infection of the supra-levator space, usually from an intra-abdominal lesion. An ano-rectal fistula can, however, be converted into a pelvi-rectal fistula by the inexperienced use of probes which can be pushed through the rectal wall searching for internal openings.

Diagnosis. The tracks of simple fistulæ can be palpated running radially from the external opening into the anal canal. The external opening can be seen and the internal opening palpated at the anal intermuscular depression. A probe passes radially along the track into the anal canal.

In complicated fistulæ the track cannot be felt except by digital examination of the anal canal when an induration can be felt on one or either side at the level of the ano-rectal ring or spreading round posteriorly under the levator ani muscle. A probe passes upwards parallel to the anal canal for 2–2½ in. and its end can be felt through the rectal wall just above the level of the ano-rectal ring. The internal opening can be felt at or above the anal intermuscular depression but below the ano-rectal ring except in pelvi-rectal fistulæ which are fortunately rare.

Treatment

General. That treatment is unsuccessful is manifest by the number of cases which present after multiple operations spread over many years.

The reason for the majority of failures is threefold: fear of dividing too much sphincter muscle and making the patient incontinent, failure to recognize clinical types of fistulæ, and failure to apply the principles of wound healing.

Healing along a track is third intention healing. It will occur spontaneously provided the pus, urine, intestinal contents, saliva or bronchial mucus can drain naturally without obstruction and provided the cause is treated

A suprapubic urinary fistula heals rapidly when urinary obstruction is relieved—so do intestinal fistulæ.

A fistula-in-ano attempts to heal but becomes periodically reinfected from the rectum and although the external opening may heal, a common story is one of "healing" and recurrent abscess formation A fistula, then, is failure of healing by third intention.

The object of treatment is to convert a track which fails to heal by third intention into a flat wound which will heal by second intention. A surgeon must be able to recognize the ano-rectal ring by touch and sight (through a proctoscope) and the anal intermuscular depression and must be aware of the clinical differences between simple and complicated fistulæ With this elementary knowledge the internal opening can be confidently located and the fear of making a patient incontinent by dividing the ano-rectal ring no longer exists

Simple Fistulæ. Under light general anæsthesia a probe is passed from the external to the internal opening and the track is laid open. Part or whole of the subcutaneous external sphincter is divided in low anal fistulæ. In high anal fistulæ variable amounts of the internal and external sphincter muscles are divided.

The wound must now be flattened, firstly by making "Salmon's back cut," a small incision outwards from the external opening, and then the sides of the wound trimmed to

produce a triangular or oval wound. The size of the external or drainage wound should be a little more than double the area of the wound in the anal canal because wounds of the anal canal take twice as long to heal as perianal wounds and the drainage wound outside the anal canal should be the last part to heal.

Complicated Fistulæ

(1) A complete ano-rectal fistula consists of a main track, two lateral extensions, and one or more internal openings. These must all be laid open.

A probe is passed up the main track and a cut is made backwards to the tip of the coccyx which is level with the apex of the ischio-rectal fossa. The probe is then passed forwards along the anterior extension on the side of the main track which is then laid open. Next, the extension to the opposite side is sought and, if present, laid open. Lastly, a careful search is made along the laid open track for internal openings; these, when identified, are also laid open.

The resulting wound extends round the whole anus except for a small strip of skin anteriorly. (The anus must never be circumcized as a chronic lymphatic œdema will result.)

The wound is now trimmed and the final result is an extensive butterfly shaped wound, extending round the back and sides of the anus.

If hæmorrhoids are present they are best removed for the extent of muscle section will result in their prolapse and thrombosis, and will embarrass wound dressing.

The majority of failures occur in this type of fistula and are due to overlooking the extension to the opposite side, missing the internal opening, or failing to recognize the possibility of two internal openings. The wound takes 3 months to heal, 6–8 weeks of which must be spent in hospital.

(2) Pelvi-rectal fistulæ, because their internal opening is above the ano-rectal ring the track cannot be laid open without making the patient incontinent. These fistulæ have therefore in the past been regarded as inoperable Healing can be achieved if the problem is tackled logically and with determination by·

(*a*) Recognizing and treating the primary pathology.

(*b*) Defunctioning the rectum with a left iliac colostomy

(*c*) Mobilizing the rectum through a posterior approach after excising the coccyx, paring away fibrous tissue round the opening into the rectum until only supple rectal wall remains and then deliberately suturing the internal opening This stage involves an operation that may last for four hours and an excessive blood loss Provision for adequate time and blood replacement up to 4–6 pints must be made

(*d*) The wound is left open to heal by granulation and at least 3 months allowed to elapse after complete wound healing before closing the colostomy

These fistulæ have been successfully treated without recourse to a preliminary colostomy

A colostomy must be recommended in reparative operations round the ano-rectum for the following reasons:

(*a*) If it is simple to make and close

(*b*) The margin between success and failure is narrow largely depending on the presence or absence of infection.

(*c*) It is more comfortable for the patient to evacuate onto the anterior abdominal

apex of the ischio-rectal fossa and can be felt above the ano-rectal ring. The internal opening (which may be multiple) is always below the ano-rectal ring.

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in simple fistulæ whose treatment presents no problem. Time saved in healing is insignificant. The proportion of failures requiring re-opening of the wound does not recommend this method.

TUMOURS

(A) Epithelial Tumours.

- (1) Benign
  - (*a*) adenoma
  - (*b*) papilloma
- (2) Malignant
  - (*a*) Primary { adenocarcinoma, squamous carcinoma, carcinoma simplex
  - (*b*) Secondary { carcinoma of cervix, carcinoma of prostate, carcinoma of colon, carcinoma of ovary, carcinoma of breast

(B) Connective Tissue Tumours.

- (1) Benign { hæmangioma, leiomyoma
- (2) Malignant { leiomyosarcoma, spindle celled sarcoma

(C) Tumours from Blood Forming and Lymphoid Tissue.

- (1) Reticulosarcoma
- (2) Lymphosarcoma
- (3) Lymphoma

(D) Miscellaneous.

- (1) Carcinoid tumours
- (2) Melanomata

Benign Epithelial Tumours

The commonest benign tumours of the rectum are adenomata and papillomata. They may be single or multiple. Their interest lies in the fact that they are liable to malignant degeneration. Twenty-five per cent of rectal carcinomata can be shown to have arisen in benign tumours, either by a small focus of carcinoma in large papillomata or the presence of a local fringe of papilloma at the margin of a large carcinoma, or the obvious intermediate stages

wall than onto a large wound behind the anus; the problem of daily wound dressing for the nursing staff is simplified.

Post-operative Care

A wound designed to heal by second intention should be covered with a FLAT moist dressing. At the time of operation a compress of 20 per cent tannic acid in 1:1,000 aqueous flavine will aid hæmostasis and help to relieve post-operative pain.

The first dressing in simple fistulæ is done on the third post-operative day following bowel evacuation by an enema. A bath is then taken and the wound irrigated with 2½ per cent Milton solution and dressed with a flat piece of gauze moistened with the same solution and a corner of gauze tucked into the anal canal.

When the surface of the wound is covered with red granulations the lotion is changed to Lotio Rubra (B.P.). Subsequently, excess granulations are controlled by silver nitrate stick and preparations applied to stimulate epithelialization. The first few dressings of complicated fistulæ with extensive wounds should be done under thiopentone anæsthesia. When the wound has set and become less painful dressings proceed as for simple fistulæ.

Each week during the healing period the wound should be examined in the theatre under anæsthesia. It is reviewed for overlooked extensions and internal openings. The wound may need to be adjusted and the patient should be warned that the operation may have to be done in stages.

General Points

A portion from the fistulous track from all surgically treated fistulæ should be sent for microscopic examination.

Continence has nothing to do with the division of the relevant sphincter muscle at right angles or the number of times it is divided. It depends on the integrity of the ano-rectal ring

Situations can arise when, owing to induration round the fistulous track and resulting rigidity of the ano-rectal ring, it is difficult to be certain of the level of the internal opening. In these cases a silk seton can be passed through the internal opening and round the sphincter muscle. The wound is drained but only a third of the muscle below the internal opening is divided. With proper drainage induration subsides and muscle elasticity returns. The level of the internal opening can then be re-assessed by palpating the silk seton in relation to an actively contracting ano-rectal ring and by visualizing the ano-rectal ring contracting over the end of a withdrawing proctoscope before the silk seton is seen; under these conditions division of the muscle surrounded by the seton is safe.

In extensive complicated fistulæ with division of the major part of the external sphincter, control of flatus is uncertain.

Ribbon gauze packed into fistulous wounds is a certain way of delaying and preventing healing. Indeed, if ribbon gauze can be packed into the wound the fistula has been inadequately laid open.

Skin grafting may accelerate the healing time of very large wounds but the addition of a large painful donor area wound is an added burden to the patient.

Many surgeons report successes of fistula excision and primary suture in attempts to promote fistula healing from second to first intention. Successes are always achieved

followed by the appearance of microscopic foci of carcinoma ("carcinoma *in situ*"). These foci coalesce to form a definite zone of carcinoma still confined to the mucosa ("focal carcinoma"). Later the stroma of the adenoma becomes invaded ("invasive carcinoma").

When a pathologist reports on a malignant adenoma three factors should be determined:

(1) Is the malignant change in the adenoma focal or invasive?

(2) Is the tumour of low, average, or high grade of malignancy?

(3) From a study of the excised adenoma is there a free margin of normal tissue proximal to the invading carcinoma?

When the malignant change is anaplastic or highly malignant an excision of the rectum must be advised at whatever stage of development the malignant adenoma may be. For malignant polyps in the stage of carcinoma *in situ* or focal carcinoma the local excision of the adenoma is adequate treatment. When the stroma is invaded the recurrence rate for local excision is between 40–50 per cent, and unless there are other contra-indications to be considered a radical excision of the rectum should be performed.



Fig 214. Malignant degeneration in adenomatous polyps in a specimen from polyposis patient. Arrows indicate malignant ulcers The number of the polyps in this rectum is small enough to have made clearance possible by diathermy if no malignant change had occurred.

Papillomata (*villous papillomata*) are much larger tumours and present in two clinical forms, either as a large soft shaggy tumour projecting into the bowel lumen like a sea anemone or a low carpet tumour creeping over the surface of the rectum. They secrete large quantities of mucus and may prolapse outside the anus and be mistaken for a rectal prolapse

These tumours undergo malignant degeneration and therefore at examination the whole surface must be carefully palpated for areas of induration Biopsy should be made from multiple areas.

Treatment. Villous papillomata present special problems in treatment. They may be removed by:

(1) Endoscopic diathermy fulguration.

(2) Local excision.

Adenomata are firm rounded tumours at first sessile and in the early stage they are usually discovered during a routine examination. Later the tumour becomes pedunculated and its surface and shape become irregular. At this stage it may prolapse outside the anus or give rise to small repeated hæmorrhages.



FIG 212 Villous papilloma, protuberant type



FIG 213 Villous papilloma, carpet type

Note upper rectum and colon have been turned inside out to show circumference of tumour.

Treatment. They should always be removed and sent for microscopic examination.

A real problem arises when a clinically benign adenoma is reported as having a focus of carcinoma. The management of malignant adenomata or polyps has been carefully investigated by Lockhart-Mummery, H E. and Dukes, C. E. (1952)

A mucus secreting adenoma first shows signs of active epithelial proliferation

and gently drawn outside the anus. The broad pedicle thus formed is then transfixed with overlapping sutures of No. 3 chromic catgut and the tumour with a margin of normal mucosa is excised with a diathermy needle. The suture line is checked for hæmorrhage and then allowed to retract into its original position. Villous papillomata as high as the upper third of the rectum may be treated in this way.

(3) CONSERVATIVE ANTERIOR RESECTION. For very large papillomata involving a large surface area of bowel some form of rectal excision is necessary. If the tumour is benign it would appear to be the ideal situation for a conservative resection. All the safety factors for a similar operation for carcinoma should be observed, especially as regards implantation and free margin, except that the free margin can be less than 5 cm.

At laparotomy palpation of the liver and regional glands may give a clue to malignant change, but it is never certain that this has or has not occurred until the pathologist has serially sectioned the growth.

(4) COMBINED EXCISION OF RECTUM. W. B. Gabriel (1952) recommends a combined excision of the rectum for large villous papillomata for the following reasons:

(1) The risk of implantation recurrence.

(2) The difficulty of defining the tumour limits by palpation at operation and the consequent risk of an incomplete removal.

(3) The fact that malignant change is common and, when present, is not localized but has already involved the regional lymphatic glands.

The risks of implantation recurrence and incomplete removal must also apply to small growths removed by local excision. Recurrences do occur after local excision, but it can rarely be determined whether the recurrence results from an implantation at the time of operation or the survival of a small focus of incompletely removed tumour.

The difficulty of determining accurately the extent of a villous tumour by palpation at examination and at laparotomy is very real. This problem is solved by operating in the lithotomy Trendelenburg position. With the patient in this position an assistant can pass a sigmoidoscope from below and visually guide the surgeon to a complete removal of the tumour.

It is difficult, however, to reconcile the sacrifice of the whole rectum for a benign tumour. When the question of malignancy is in doubt the focus of carcinoma is so small that a conservative resection is surely safe.

Polyposis Intestini. The rectum is invariably involved in this hereditary disease. Multiple adenomatous polyps are studded over the surface of the rectum in varying degrees of density. They may be so closely packed that no normal mucosa can be seen. Carcinomatous change is inevitable and early (average age 35 years).

The logical treatment of this disease is to remove the whole colon and rectum. Patients, however, present for treatment young; the average age for onset of symptoms is about 20 years. The symptoms are mild and the recommendation of a permanent ileostomy is not well received.

As a compromise, and where rectal polyps are not too numerous, they may be destroyed by diathermy and a subsequent ileo-rectal anastomosis performed; the patient must be prepared for a regular life-long check on the rectal stump.

Polyps can be too numerous to be removed by diathermy without stricture formation. Attempts have been made in these cases to "core out" the rectal mucosa and line the remaining muscular rectum with ileum (ileo-anal anastomosis). The functional result,

(3) Conservative anterior resection of rectum.

(4) Combined excision of the rectum.

(1) ENDOSCOPIC DIATHERMY FULGURATION. The tumour is systematically destroyed by the diathermy button. The area should not involve more than half the circumference of the bowel wall; one or several treatment sessions may be required. The tumour



FIG 215. Densely packed polyps too numerous to be cleared by diathermy

destruction is followed by scarring and if too large a tumour is treated in this way a rectal stricture may develop

Great care must be taken that all the tumour is destroyed and a close, careful follow-up is essential Unless treatment is thorough diathermy can possibly precipitate malignant change

(2) LOCAL EXCISION Many rectal papillomata up to 1½–2 in in diameter can be prolapsed outside the rectum and drawn down on a pseudo pedicle. This is always possible for tumours which have prolapsed spontaneously. Following preliminary stretching of the anus the tumour is grasped with one or more sponge holding forceps

at night and is passed when the patient rises in the morning, being interpreted as morning diarrhœa, or it may be noticed as a constantly recurring soiling of underclothes.

(5) Urinary Symptoms. When it is remembered that at an early stage of development the rectum and urethra are represented by a common cloaca, it is not surprising that disease in one will produce symptoms in the other. Frequency of micturition, dysuria, and the passing of matter from the rectum when passing urine are not infrequent symptoms. In advanced rectal growths, size may cause urinary obstruction and retention and actual involvement of the bladder, pyuria, and pneumaturia.

Symptoms due to the complications of a rectal growth are those of:

(1) Intestinal Obstruction—unaccustomed flatulence, intermittent distension, lower abdominal ache, and mild colic are signs that the bowel is becoming obstructed—these are symptoms of the constricting type of growth occurring as a rule in the upper third of the rectum or rectosigmoid region.

(2) Shortness of Breath, lassitude, anorexia, and loss of weight are symptoms of secondary anæmia and the specific effect of a cancer on its host.

(3) Symptoms of Widespread Disease. Pain in the right upper abdominal quadrant suggests the presence of liver secondaries, pain in the chest and dyspnœa the presence of lung secondaries with pleural effusion.

Signs. If a patient complains of rectal symptoms or an alteration in the usual rectal function it hardly seems necessary to advise making a digital examination of the rectum, yet this examination is still omitted or postponed to another visit or consultation. It should be done at once.

On inserting a finger into the rectum of a patient with a rectal carcinoma, something abnormal will be felt. No rectal carcinoma is out of reach of the average length finger —if the perineum is pressed up when the finger is fully inserted.

Very occasionally, the combination of an obese patient with large buttocks and a short examining finger may mean that growths of the upper third or rectosigmoid are difficult to palpate.

While the finger is in the rectum the free hand should palpate the lower abdomen and the pelvis be thoroughly explored by bidigital examination—growths of the lower sigmoid colon are often diagnosed in this way when other methods have failed to detect them.

The abnormal physical sign detected by palpation will depend on the gross characteristics of the carcinoma which may be one of the following types:

(1) Ulcerating growth.
(2) Protuberant growth
(3) Constricting growth.
(4) Carcinoma arising in an adenoma or papilloma.
(5) Atypical type.

(1) Ulcerating Carcinoma. The usual ulcer crater is hard and has raised, everted edges. The ulcer may be a deep small ulcer penetrating the rectal wall and extra-rectal tissues with no raised edge—this type of ulcer is highly malignant and, although small. has a bad prognosis.

(2) Protuberant Carcinoma (hypertrophic or "cauliflower"). This type grows into the rectal lumen as an irregular dome-shaped swelling and the prognosis is comparatively favourable.

however, is so indifferent and motions so frequent that a permanent ileostomy is preferable.

References

Gabriel, W. B (1948) The Principles and Practice of Rectal Surgery, 4th edition, Lewis, London, p. 330 (hæmangiomas).
Gabriel, W. B. (1952) *Proc. Roy. Med. Soc.* 4., 685 (Combined excision).
Lockhart-Mummery, H E. and Dukes, C. E. (1952) *Lancet*, 2, 751 (adenomata).

RECTUM AND ANAL CANAL CARCINOMA

Introduction

Cancer of the rectum is one of the common cancers. The diagnosis can be made by palpation without any special aid. The prognosis, when treated in time and efficiently, is better than that of any malignant growth elsewhere in the body. If, therefore, the symptoms are recognized, a finger inserted into the rectum, and sound principles of treatment based on pathology applied to its eradication, the opportunity exists to treat an unpleasant and lethal disease successfully.

Symptoms. Occasional instances occur when an unsuspected rectal growth is fortunately discovered during routine examination. The first episode in the story of a rectal cancer is the awareness and reporting of symptoms by the patient. These symptoms are those caused by the growth itself and those resulting from its complications, and vary according to the type of the growth and its position in the rectum.

As the growth is usually in ulcer form with destruction of epithelium, bleeding and rectal irritation must result, to give rise to one or a combination of the following symptoms:

(1) Bleeding.
(2) Alteration in bowel habit.
(3) Pain and tenesmus.
(4) Discharge
(5) Urinary symptoms

(1) Bleeding. Man, in common with many other animals, inspects his excreta. The appearance of blood smeared on or intermingled with fæces is an early sign, unfortunately frequently dismissed as piles by the patient. Occasionally a large hæmorrhage may occur as a first symptom. When bleeding is noticed in between or apart from defæcation it is of diagnostic significance. In summer, when salads are on the menu, unmasticated undigested beetroot appears in the stool and is not infrequently mistaken for blood

(2) Alteration in Bowel Habit Diarrhœa, frequency, and a feeling of incomplete evacuation are common with growths of the rectal ampulla Urgency of defæcation progressing to incontinence are symptoms of growths of the lower third and anal canal.

(3) Pain and Tenesmus The anal canal is richly supplied with sensory nerve endings; pain and abortive painful straining indicate a growth of the anal canal or growths invading the anal canal from above

(4) Discharge. This consists of altered blood, mucus produced by the irritated columnar rectal epithelium, and liquid fæces The discharge accumulates in the rectum

There is no ulceration of the surface of the rectum and it is of a rubbery, resilient feel, as if the rectum has been converted into fibrocartilage.

(4) Carcinoma Arising in an Adenoma or Papilloma. One quarter of rectal carcinomas can be demonstrated to have arisen in benign tumours. A far larger percentage undoubtedly start as a benign growth which is completely destroyed by its malignant change.

Villous tumours, especially the low carpet type, are not easy to palpate. They are soft and differ only slightly in consistency from the rectal wall but must be palpated thoroughly; localized areas of induration are diagnostic of malignant degeneration.

Very small adenomata may contain a focus of carcinoma and present as a simple polyp. All polyps and adenomata when recognized should therefore be removed and examined microscopically.

(5) Atypical Types. These types are notoriously hard to diagnose and their recognition depends on the clinical acumen of the doctor and his knowledge that such forms exist: they present in various ways—as a slight submucous induration—as a fistula—as a small erosive ulcer very similar to a fissure-in-ano—or as an induration on a prolapsed pile. The tubular constricting and deeply penetrating ulcer type are also atypical forms of rectal growth.

The appreciation by palpation that there is a growth in the rectum does not end the examination The site of the growth in the rectum, the quadrants of the bowel involved, the mobility of the growth and its involvement of related viscera should all be determined and recorded.

Glands can often be palpated in the mesorectum above the growth through the posterior rectal wall.

In women a vaginal examination must be made to assess involvement of the recto-vaginal septum.

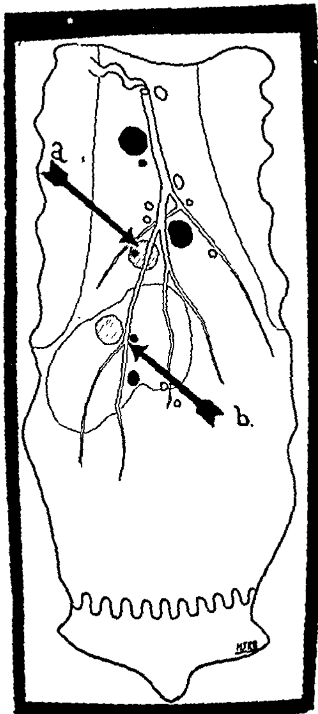


Fig 217. Gland dissection from Fig 216.
(a) Level of malignant adenoma.
(b) Villous papilloma—shaded area indicating malignant degeneration Invaded glands are filled in in black

(3) Constricting Carcinoma. Sometimes advanced growths of the anal canal or lower third of the rectum so constrict the lumen as to make the passage of a finger through the growth extremely painful. In these circumstances the examination should be repeated under anæsthesia.

Constricting growths also occur in the rectosigmoid and upper third of the rectum. Intestinal obstruction may be the first symptom

A diffuse, tubular stenosis of the rectum is caused sometimes by anaplastic growths.

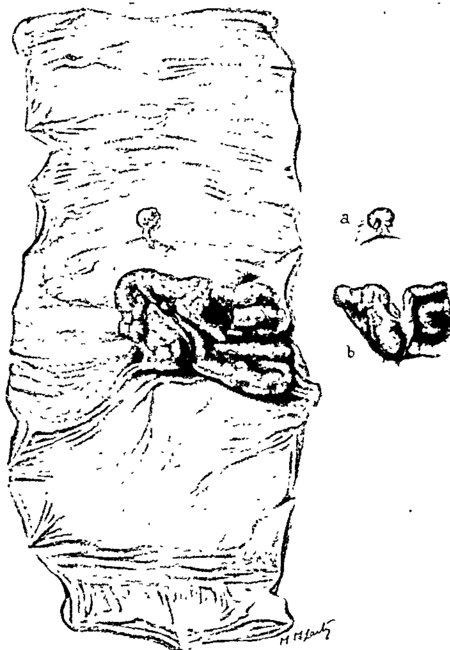


Fig 216 Malignant degeneration in an adenoma and villous papilloma in the same specimen

(a) Malignant adenoma

(b) Focus of malignancy in villous papilloma

Diagnostic Aids. The only special local examinations of value are proctoscopy and sigmoidoscopy to visualize the growth, and the removal of a piece for microscopic examination or biopsy. Convenient forceps for a biopsy through a proctoscope are Caldwell-Luc forceps or a Hartmann's conchotone. Longer forceps are required for biopsy through a sigmoidoscope, Patterson's forceps being a very convenient pattern.

It is bad practice to pass a sigmoidoscope above the growth. Many tumours at time of treatment have growth already in the lumen of the tributaries of the hæmorrhoidal veins. Squeezing of the tumour between a sigmoidoscope and the boney pelvic walls is one certain way of despatching growth emboli from the lumen of a vein to the liver. It is true that additional lesions and second primary tumours often co-exist with a rectal cancer—these can wait until laparotomy for identification.

In making the biopsy a portion of the ulcer edge should be removed. Difficulties in obtaining suitable material arise in ulcers with no edge and atypical forms of tumour, such as the tubular stricture. In these cases the biopsy is better taken under anæsthesia, when the growth can be generally re-assessed. The value of the biopsy lies in confirming the clinical diagnosis, determining the histological type, and grading the tumour according to its degree of malignancy. It is customary to make a cytological classification of growths into low, average, and high grade of malignancy, according to whether the cell is atypical or not. The biopsy can influence the plan for treatment; for instance, a high grade growth or a colloid growth is quite unsuitable for an operation designed to retain the anal sphincters.

Differential Diagnosis

So common are the pitfalls of diagnosis that it is prudent to wait for a confirmatory biopsy report before excising a rectum for malignant disease. A surgeon making this a hard and fast rule is unlikely to make the mistake of excising the rectum for a benign lesion and if the malignancy of the biopsy specimen is graded valuable additional information is obtained on which to base and modify treatment

Most of the diseases listed below are uncommon but successful diagnosis depends on a knowledge of the possibility of the unusual.

The following conditions have been mistaken for and many treated as a carcinoma of the rectum:

(1) Foreign Body Granuloma. Foreign bodies such as game or rabbit bones can become impacted across the rectal lumen forming an inflammatory tumour clinically indistinguishable from a rectal neoplasm.

(2) Paraffinoma. The injection of hæmorrhoids with unabsorbable oils can produce a chronic persistent ulcerated tumour

(3) Amœbic Granuloma. R. W Nevin (1949), describing rectal lesions in amœbiasis as pin point lesions, large, single or multiple ulcers, crateriform pits or granulomata, states that they are easily confused with a rectal carcinoma.

(4) Bilharzial Granuloma.

(5) Tuberculous Ulceration. An early tuberculous rectal ulcer can be indistinguishable, except microscopically, from an early, flat, carcinomatous ulcer.

(6) Granulomatous (Syphilitic) Ulceration of the Rectum.

(7) Actinomycosis can involve the rectum primarily or secondarily The ampulla is commonly involved in the primary cases. Secondary involvement results from disease

It assists in the assessment of mobility if the patient strains down during the examination

In the general examination of the patient, the inguinal glands, the glands in the left side of the neck, the liver, and the state of distension of the colon should be noted. Fæcal lumps accumulate in the pelvic colon and there is often a tell-tale distension of the cæcum.

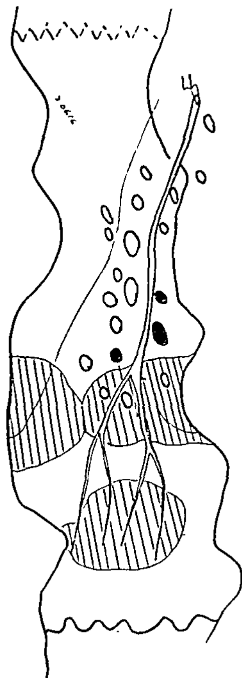


FIG 218 Diagram to show method of embolic spread to liver.



FIG. 219. Additional lesion and second primary tumour in carcinoma of rectum.

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elsewhere in the abdomen, such as the appendix, ileocæcal region, stomach or ovaries, Zachary Cope (1949).

(8) ENDOMETRIOSIS may involve the upper third of the rectum and form a tumour mass protruding into and obstructing the rectal lumen.

(9) LYMPHOGRANULOMA.

(10) RECTAL STRICTURES. The diagnosis of a rectal stricture due to an atypical anaplastic carcinoma, from a rectal stricture secondary to carcinoma of the prostate or from chronic inflammatory lesions, is both a difficult clinical and histological problem.

(11) CARCINOMA from the colon, ovary, and prostate may invade and ulcerate into the rectum to simulate a rectal carcinoma.

(12) ATYPICAL forms of ano-rectal carcinoma can masquerade as most of the common forms of minor rectal lesions. A small erosive anal carcinoma can simulate a fissure-in-ano, a melanoma a thrombosed pile, and a burrowing colloid carcinoma a fistula-in-ano.

(13) SARCOMATA are usually globular, protruding tumours covered to a variable degree by rectal mucosa.

References

Nevin, R. W. (1949) *Proc. Roy. Soc. Med.* 42, 769.
Zachary Cope, V. (1949) *Proc. Roy. Soc. Med.* 42, 763.

PATHOLOGY OF RECTAL CANCER

Location of Rectal Carcinomas

For convenience of description and for purposes of localization of rectal cancer, the rectum may be divided into lower, middle, and upper thirds and a rectosigmoid zone. This zone is above the limits of the anatomical rectum (third sacral vertebra) but at or below the sacral promontory.

Ampullary growths are at or below the peritoneal reflection and their lower edge is seen at 8 cm from the anal margin. Upper third growths are above the peritoneal reflection and their lower edge is seen at 12–15 cm. from the anal margin. Rectosigmoid growths are sited at or just below the sacral promontory and their lower edge is seen 15–20 cm. from the anal margin.

Approximately 37 per cent of growths occur in the lower third, 32 per cent of growths in the ampulla and 31 per cent of growths in the upper third and rectosigmoid zone.

There is a difference in prognosis in growths of the lower and middle thirds and growths of the upper third and rectosigmoid zone or growths below and above the peritoneal reflection Growths below the peritoneal reflection with their close relationship to the prostate, urethra, and vagina and their proximity to the lateral pelvic walls and lateral ligament attachment have a considerably lower five year survival rate than growths above the peritoneal reflection.

Histological Types

The rectum and anal canal are lined by columnar, transitional, and squamous epithelium and growths arise with corresponding histological characteristics from each of these epithelia.

Most malignant growths are columnar celled adenocarcinomas. They account for

95 per cent of growths. Squamous and transitional celled carcinomas account for only 5 per cent of growths.

Adenocarcinomas may secrete mucus (mucus secreting carcinoma) and are commonly known as colloid carcinomas. The mucus distends the cell, altering its shape, and compressing the nucleus against the cell wall to give rise to the characteristic "signet" cell.

Colloid growths spread more rapidly and metastasize earlier than adenocarcinomas. They present in atypical forms and spread by unusual channels. These characteristics make the prognosis worse in colloid growths compared with adenocarcinoma.

Squamous and transitional celled carcinomas arise in the anal canal and are complicated by their possible lymphatic spread to the inguinal glands.



FIG. 220 Colloid carcinoma.

Occasionally mixed tumours occur in the ano-rectal region. They may be composed partly of squamous and partly of columnar epithelium. Their lymphatic metastases have a similar mixed pathology.

Classifications. Two classifications are of great value in the management of ano-rectal cancer:

(1) Broders' classification based on cell differentiation.

(2) Dukes' classification based on the extent of spread of the disease demonstrated at dissection of the operation specimen.

(1) *Broders' classification.* Tumours are classified into four grades. In grade 1 the majority of cells are well differentiated, in grade 4 the majority of cells are undifferentiated, grades 2 and 3 being intermediate stages. A useful modification of this classification is to subdivide adenocarcinomas according to the degree of differentiation of the tumour cells into low grade, average grade, or high grade of malignancy.

Low grade tumours consist of well differentiated cells with almost normal regular acini.

Average grade tumours have recognizable columnar cells and a definite but irregular and distorted acinar formation.

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Most malignant growths are columnar celled adenocarcinomas. They account for

High grade tumours are composed of polygonal or spheroidal cells with no glandular arrangement.

This classification has practical application both before and after the tumour is removed. The biopsy should be thus graded. If a clinically fixed advanced growth is reported on as being of low grade of malignancy it is an encouragement to persist with a radical excision as in all probability fixation is largely due to sepsis; if, on the other hand, the growth is reported as being of high grade of malignancy, then nothing but the most radical operation possible offers any hope of a cure, however small the growth may be.

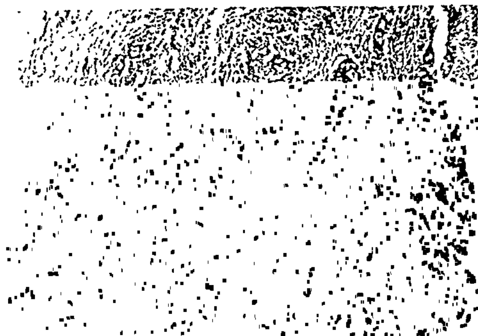


FIG 223 Adenocarcinoma, high grade

A final assessment of malignancy is made on the removed tumour. This may occasionally differ from the biopsy report and indeed different areas of the same tumour vary in degree of malignancy.

The value of the histological grading after removal of the tumour lies in determining prognosis.

(2) *Dukes' classification* is based on the extent of spread of the growth as demonstrated in the operation specimen

"A" case—the growth is limited to the rectal wall

"B" case—the growth has spread into the perirectal tissues, but with no lymphatic metastases.

"C" case—the growth has involved the lymphatic glands.

Because of the wide variations in degree of lymphatic gland spread and the corresponding difference in prognosis, "C" cases are subdivided into "C_1" and "C_2" cases. In "C_1" cases there are metastatic deposits in the glands adjacent to the tumour but glands free from growth can be demonstrated below the upper limit of the excised vascular pedicle In "C_2" cases the highest gland removed contains metastatic deposits.

Dukes' classification has an important application in determining prognosis.



FIG 221 Adenocarcinoma, low grade.



FIG 222 Adenocarcinoma, average grade.

High grade tumours are composed of polygonal or spheroidal cells with no glandular arrangement.

This classification has practical application both before and after the tumour is removed. The biopsy should be thus graded. If a clinically fixed advanced growth is reported on as being of low grade of malignancy it is an encouragement to persist with a radical excision as in all probability fixation is largely due to sepsis; if, on the other hand, the growth is reported as being of high grade of malignancy, then nothing but the most radical operation possible offers any hope of a cure, however small the growth may be.

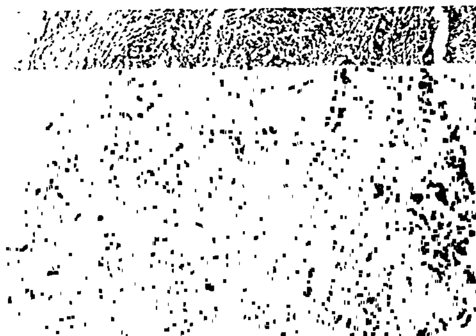


FIG. 223. Adenocarcinoma, high grade

A final assessment of malignancy is made on the removed tumour. This may occasionally differ from the biopsy report and indeed different areas of the same tumour vary in degree of malignancy.

The value of the histological grading after removal of the tumour lies in determining prognosis.

(2) *Dukes' classification* is based on the extent of spread of the growth as demonstrated in the operation specimen

"A" case—the growth is limited to the rectal wall

"B" case—the growth has spread into the perirectal tissues, but with no lymphatic metastases

"C" case—the growth has involved the lymphatic glands

Because of the wide variations in degree of lymphatic gland spread and the corresponding difference in prognosis, "C" cases are subdivided into "C_1" and "C_2" cases. In "C_1" cases there are metastatic deposits in the glands adjacent to the tumour but glands free from growth can be demonstrated below the upper limit of the excised vascular pedicle. In "C_2" cases the highest gland removed contains metastatic deposits.

Dukes' classification has an important application in determining prognosis.



FIG. 221 Adenocarcinoma, low grade.



FIG 222 Adenocarcinoma, average grade

Occasionally, however, the glands nearest the tumour are free from growth but a metastasis is found in a gland high up on the inferior hæmorrhoidal chain of glands. This is known as discontinuous upward spread and is an unfavourable prognostic feature.

Lymphatic spread occurs outwards along the lateral ligaments and over the fascia on the levator ani muscle to involve glands of the pelvic wall.

Invasion of the lymphatic glands below the primary tumour only occurs in high grade growths and very advanced growths where the lymphatic glands above are already full of growth.

Rectal growths may also metastasize to the pre-aortic glands, to glands round the cœliac axis and cisterna chyli and to the left supraclavicular fossa. Permeation of lymphatic vessels is a feature of anaplastic growths and is a method by which growth may spread below a tumour.

(c) Venous Spread. Growth can be detected in the tributaries of the inferior hæmorrhoidal veins. The reported incidence varies from 15–50 per cent and obviously depends on the methods employed to detect intravenous growth and on their thoroughness. Its extent may vary from a small plug of growth in a venous tributary emerging from the primary tumour to massive involvement of the whole inferior hæmorrhoidal and mesenteric veins.

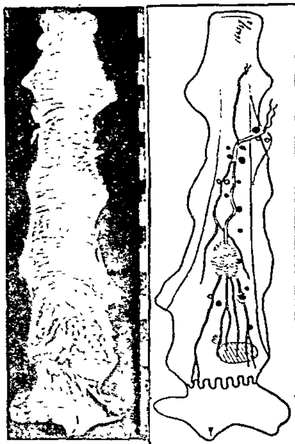


Fig. 225 Small early adenocarcinoma average

Fragments of growth become dislodged and pass up the portal vein to the liver to give rise to hepatic metastases.

There are two practical considerations arising from invasion of veins and venous growth emboli.

Firstly, the passage of a sigmoidoscope past or through a rectal growth and its compression against the pelvic wall may dislodge growth emboli. A sigmoidoscopic examination should not be pressed beyond the lower edge of the tumour.

Secondly, at operations for excision of a rectal cancer, the inferior mesenteric vein must be ligated before the growth is mobilized, to avoid the risk of dislodging venous emboli into the portal circulation.

Venous spread may occur in submucous veins at some distance from the edge of the primary tumour and the growth may then erupt to the surface to simulate a second primary growth.

Spread downwards, retrograde venous spread, has also been demonstrated.

After a radical excision 90–100 per cent of five year cures can be expected in "A" cases, 60 per cent of five year cures in "B" cases, and 25 per cent of five year cures in "C" cases.

In a *series of rectal carcinomas* a surgeon may expect to find growths in approximately the following percentages in each classification:

	Per cent		*Per cent*
Low grade	10	"A" case	15
Average grade	65	"B" case	35
High grade	25	"C" case	50

An obvious relationship exists between histological types and extent of spread. The majority of high grade malignancy growths are "C" cases and low grade malignancy growths are "A" cases.

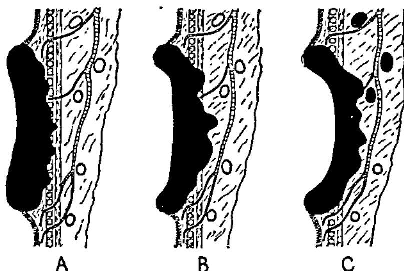


FIG. 224. Dukes' classification based on the extent of spread of the disease demonstrated at dissection of the operation specimen
A. Growth limited to wall of rectum B. Extension of growth to extrarectal tissues but no metastases in regional lymph nodes. C Metastases in regional lymph nodes

Methods of Spread

(*a*) LOCAL SPREAD. A rectal carcinoma spreads over the surface of the rectum in upward, downward, and lateral directions to produce an oval ulcer averaging 2–3 in. in diameter at time of operation. It spreads more readily in a transverse than in a longitudinal direction.

Direct spread by submucous infiltration well beyond the margins of the primary tumour can sometimes be demonstrated and is a common feature of colloid growths

It takes six months for an average growth to involve one quarter of the bowel circumference. The growth also spreads through the layers of the bowel wall to invade the perirectal fascia and fat and adjacent viscera.

Direct involvement of adjacent viscera may result in a rectovaginal fistula in the female, a recto-urethral fistula or a rectovesical fistula in the male.

(*b*) LYMPHATIC SPREAD. This occurs in an upwards direction from the primary tumour along the inferior hæmorrhoidal vessels. The glands adjacent to the growth are invaded first, subsequent glands becoming involved in a sequence from below upwards

Occasionally, however, the glands nearest the tumour are free from growth but a metastasis is found in a gland high up on the inferior hæmorrhoidal chain of glands. This is known as discontinuous upward spread and is an unfavourable prognostic feature.

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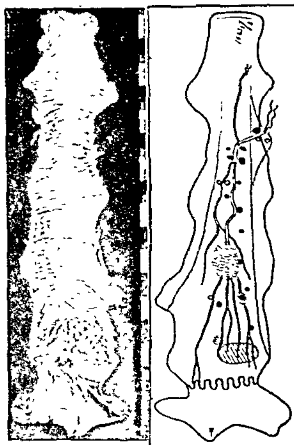


Fig. 225. Small early adenocarcinoma average grade in a male aged 34, removed by extended combined excision. Note widespread lymphatic metastases Death occurred in 2 years 8 months from generalized carcinomatosis

There are two practical considerations arising from invasion of veins and venous growth emboli.

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Venous spread may occur in submucous veins at some distance from the edge of the primary tumour and the growth may then erupt to the surface to simulate a second primary growth.

Spread downwards, retrograde venous spread, has also been demonstrated.

(*d*) Arterial Spread Occasionally in cases with widespread intra abdominal secondaries multiple growth nodules are seen along the mesenteric border of the small intestine. The probable explanation is that a branch of the superior mesenteric artery has been invaded by growth and arterial emboli are showered off to the periphery.

(*e*) Transperitoneal Spread in its early form may be seen as a plaque-like thickening in the peritoneum continuous with peritoneal puckering over the primary tumour or as small pin head nodules round the growth. Transperitoneal spread takes place by way of subserous lymphatics or cell dissemination by ascitic fluid.

(*f*) Transplantation. Rectal growths can be transplanted onto granulating or wound surfaces. The practical application of this knowledge can be seen in various ways:

(1) Recurrences have been reported in abdominal wounds and round colostomies following excision of the rectum.

(2) A growth high up in the rectum can transplant onto a fistulous track at the anal margin.

(3) Recurrences following conservative resections are due to transplantation of growth fragments from the bowel lumen to the suture line.

(4) At operation a surgeon should handle a growth as little as possible and, if practical, cover the growth surface with gauze moistened with 1:500 mercury perchloride.

Miscellaneous

In common with other malignant tumours rectal growths may metastasize throughout the body, common sites for metastases being the lungs and brain. As part of the pre-operative examination of a patient with rectal cancer, the chest should be X-rayed. Very occasionally, the detection of small secondaries in the lung affords the opportunity for their removal by lobectomy or pneumonectomy.

TREATMENT OF CARCINOMA OF THE RECTUM

General

No other form of treatment yet offers as good a chance of cure as a radical excision. Surgery can give a 45–50 per cent five year cure.

The aim of a radical excision is to remove all macroscopic growth and tissue zones where spread may take place.

It may be achieved by a combined excision of the rectum where the rectum, its lateral ligaments and mesentery are removed *en bloc* and the patient left with a permanent colostomy.

Ever since excision of the rectum for carcinoma has been practised attempts have been made to preserve the sphincter ani and normal function. Anterior or abdomino-anal resection where continuity of the bowel is restored can offer an equal chance of cure to selected cases of carcinoma of the rectum.

A surgeon is therefore faced with the problem of deciding how to select cases for a combined excision or a restorative resection. The only difference in the extent of the operation is leaving a small rectal stump in restorative resections. The remainder of the operation is in no way less radical than a combined excision. The problem, therefore, is

one of knowing if, when, and how often the rectal stump can be invaded with growth, or under what circumstances downward spread of the growth occurs.

Downward spread of growth can occur by:

(1) Lymphatic gland spread.

(2) Submucous infiltration.

(3) Lymphatic permeation.

(4) Retrograde venous spread in continuity or by embolus.

Seventy-five per cent of specimens showing downwards spread occur in highly malignant and colloid growths—almost all the remainder in very extensive growths when the upward lymphatic drainage is already blocked by growth. Downward spread rarely occurs 5 cm. below the lower edge of the growth.

If highly malignant, atypical and colloid growths and very extensive growths are not selected for restorative excision and if when a restorative excision is done the bowel is transected at least 5 cm. below the lower edge of the growth, then there is little likelihood of growth being left in the rectal stump.

In spite of this selection there remains the fact that recurrences occur in the anastomotic zone of restorative resections and this is due to failure of surgical technique.

Clumps of cancer cells break off from the parent tumour and can be demonstrated in centrifuged rectal washings to have been lying loose in the bowel lumen These cells can be implanted on the anastomotic suture line unless thorough steps are taken to remove them before the anastomosis is made.

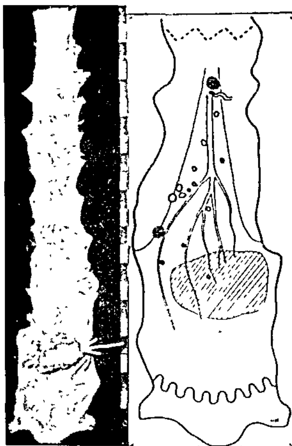


FIG 226 Discontinuous upward lymphatic spread.

The ideal criteria for a restorative resection are a thin female patient with a long pelvic colon and an early mobile growth of low or average grade of malignancy situated at least 10 cm from the anal margin The prognosis of young patients with carcinoma of the rectum is so poor that nothing but the most extensive and radical excision should be contemplated.

In considering treatment for a carcinoma of the rectum the surgeon should always explore the possibility of a restorative resection. It will be found that this cannot be done in lower third and most middle third growths. It can be done in the majority of growths of the upper third and rectosigmoid. Highly malignant, colloid, and very extensive growths being excluded, the possibility of saving the patient a permanent colostomy exists in about 25 per cent of cases.

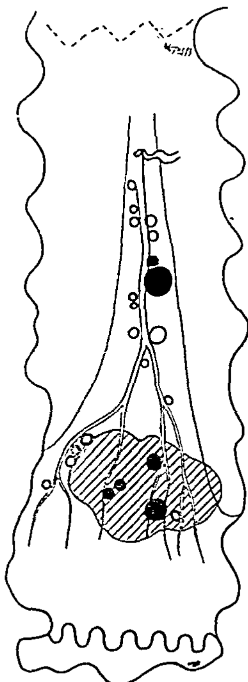
Assessment of Operability

There are only two contra-indications to the removal of a rectal growth: very severe cardiac or renal disease and extensive widespread metastatic growth.

Medicine has progressed so far in the last twenty years that major surgery is possible in the presence of most constitutional diseases.



(a)



(b)

FIG. 227 (a and b). Adenocarcinoma in a girl of 17 and gland dissection. In spite of radical excision death occurred in 4 years 4 months from lung metastases.

The advice of a physician who is familiar with the proposed operation should be obtained. He can assist in preparing the patient, assessing the risks, and guiding the post-operative treatment. Should post-operative heart and lung complications arise, he is at an advantage if he has examined the patient before operation.

Old age (octogenarians) is no contra-indication—in fact, old people stand major surgery under modern conditions very well.

Obesity carries great penalties: it makes the surgeon's task most difficult, it limits the extent of the operation, wound complications are common, and a fatty infiltrated

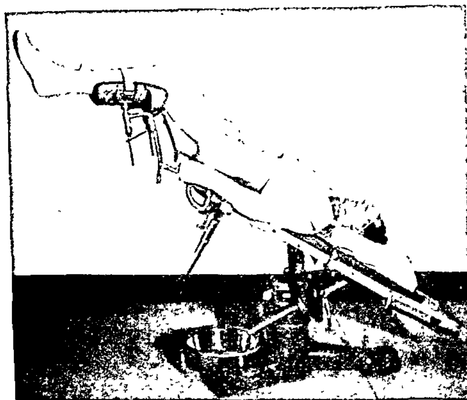


FIG 228 Abdominal view of patient in lithotomy-Trendelenburg position

heart cannot respond so well to additional strain. One or two months' postponement of the operation in order to reduce weight, while undesirable from the cancer aspect, may reduce the operative risk in these cases.

A growth should not be judged inoperable from local fixation assessed clinically. Many apparently fixed growths mobilize well at operation—and only after laparotomy and a failed trial dissection should a growth be deemed inoperable from local fixity.

Combined Excision of the Rectum

A combined excision of the rectum can be practised in three ways:

(1) Abdomino-perineal excision (Miles).

(2) Perineo-abdominal excision (Grey Turner and Gabriel).

(3) Synchronous combined excision (Lloyd-Davies).

In the abdomino-perineal excision the rectum is dissected out from the abdomen with the patient in the Trendelenburg position and the perineal excision left to the end of

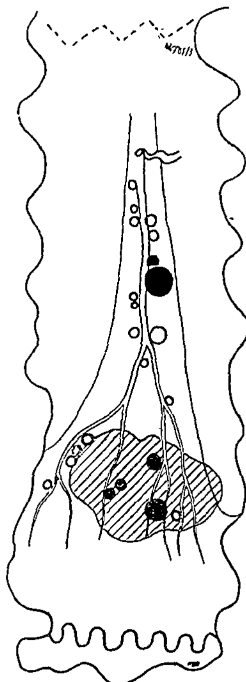
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(a)



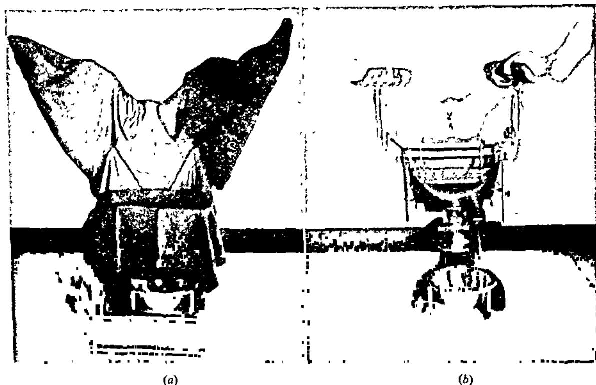
(b)

FIG 227 (*a* and *b*) Adenocarcinoma in a girl of 17 and gland dissection In spite of radical excision death occurred in 4 years 4 months from lung metastases.

or a resection can be made until after laparotomy, the site of the colostomy is marked by skin scratches.

(2) A left paramedian incision from above the umbilicus to the symphysis gives the best access to the operative field.

(3) The abdomen should be explored methodically and carefully. The liver and gall bladder are palpated first, then the colon to exclude second primary growths and other pathology (diverticula). The remaining viscera are checked, especially the omentum for secondary deposits. Finally, the growth itself is identified, assessed for mobility, and



(a) (b)

FIG 230 Perineal view of patient in lithotomy-Trendelenburg position.
(a) "Towelled-up" with instrument table in place
(b) Not "towelled up"

relative measurements to the peritoneal reflection and sacral promontory taken. The mesorectum is palpated for glands, and surrounding peritoneum for growth nodules.

(4) The next step, before mobilizing the growth, is the ligation of the inferior mesenteric vessels. The tie in the standard operation is usually just below the left colic or first sigmoid arteries. Venous invasion of the tributaries of the inferior hæmorrhoidal vein has been found in as many as 50 per cent of operative specimens. If the growth is mobilized before tying the inferior mesenteric vein there is great risk of growth emboli being despatched to the liver

(5) The rectum is next mobilized by freeing it posteriorly and then anteriorly between the vesicles and prostate in the male and the vagina in the female.

(6) The lateral ligaments are then divided as close to the pelvic wall as possible.

(7) The levator ani and post-rectal fascia are divided from the perineum.

(8) *Adherent viscera.* Where possible, these should be in part or wholly excised with the rectum Loops of small intestine, portions of bladder, uterus tubes, and ovaries are

the operation. In the perineo-abdominal excision the rectum is dissected out from the perineum with the patient in the left lateral position and the operation finished in the abdomen. *These operations can be done single-handed by one surgeon.*

The synchronous combined excision is performed in the lithotomy-Trendelenburg position by two surgeons working simultaneously at the perineum and abdomen. Its advantages are that the operating time is shortened, and very extensive, fixed growths can be removed with greater ease and safety.

The lithotomy-Trendelenburg position, Lloyd-Davies (1939), has many advantages. An excision can be carried out single-handed without altering the patient's position

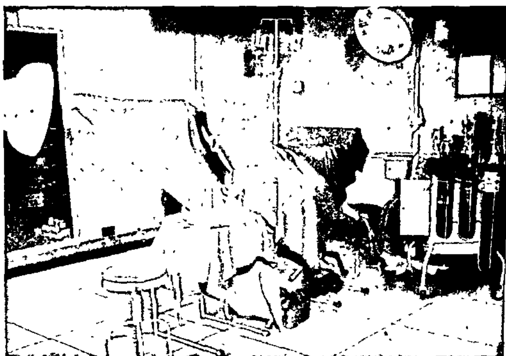


FIG 229 A composite view of lithotomy-Trendelenburg position before start of operation Note position of accessory lights Both operative fields must be efficiently illuminated Disappointment in the advantages claimed for this position often results from failure to study this elementary but essential point in theatre technique

during the operation. Two surgeons can work at the same time and in extensive fixed growths this increases the resectability rate and makes an otherwise difficult operation relatively easy. Anterior resections can be performed with equal ease and the key step in this operation, of washing out the rectal stump, can be done without disturbance. In very low resections the anal cuff can be everted and an abdomino-anal anastomosis performed outside the anus by an assistant or second surgeon.

Steps of a Combined Excision

(1) The colostomy is an end colostomy It should be sited over the outer third of the left rectus muscle, just below the umbilicus The colostomy opening in the abdominal wall is best made as the first step in the operation, after the abdominal incision is made the layers of the abdominal wall retract on each other. If it is not certain if an excision

(4) Measurements obtained on the excised specimen after fixation.

(5) The distance of the anastomotic line from the anal margin assessed at examination in the follow-up period.

The majority of anterior resections can be managed as one stage operations.

Abdomino-anal excision should always be performed as a two or three stage operation, i.e. a right transverse colostomy at the time the growth is resected or a similar colostomy performed one week before the growth is resected, the resection and subsequent colostomy closure.

The question of staging the operation depends on how well it has been possible to prepare the bowel and whether there is an adequate blood supply to the anastomosis with no tension at the suture line.

Anterior Resection

The steps of this operation are as follows:

(1) The most convenient position is the lithotomy Trendelenburg position. It allows the rectal stump to be irrigated and cleaned without moving the patient. First class lighting facilities, diathermy, and suction appliances are essential equipment.

(2) The best approach is through a long left paramedian incision.

(3) A standard exploration of the abdomen is made.

(4) The splenic flexure is mobilized; the inferior mesenteric artery and vein are ligated.

(5) The growth is mobilized posteriorly and in front and the lateral ligaments divided.

(6) The vascular pedicle at the back of the rectum is divided.

(7) A right-angled clamp is placed across the rectum at least 5 cm. below the lower edge of the growth and the rectal stump is now carefully irrigated with 1/500 mercury perchloride solution. It must be re-emphasized that this is a vital step in the operation

(8) Two stay sutures are now placed at the two corners of the rectum which is now divided below the clamp. The upper division of the colon is now made to provide a length of bowel which is both viable and long enough to be anastomosed without tension.

(9) A two-layer end-to-end anastomosis joins the two ends together and the retro-anastomotic area is drained with corrugated rubber covered by Paul's tubing and laid retroperitoneally through the stab drain in the left inguinal region.

Abdomino-anal Excision

The preliminary steps of an abdomino-anal excision are identical with those of an anterior resection, the only exception being that an additional length of colon will be required to effect the anastomosis which is made by everting the lower rectal cuff through the anus and suturing the colon to the rectal stump as in a rectosigmoidectomy.

Complications Arising in a Patient Undergoing an Excision of the Rectum

These complications are unfortunately numerous and in few other abdominal operations is there such a need for careful preparation, practised surgery, and vigilant post-operative care.

easily removed. Involvement of the prostate and urethra in the male makes cure only possible by a pelvic clearance.

It must be emphasized that even very light adhesions to another viscus may have already spread cancer cells beyond the rectum. The posterior vaginal wall should be removed if there is any possibility of the recto-vaginal septum being involved.

Extended Combined Excision

The standard combined excision of the rectum as practised today does not include in its scope all the possible zones of cancer spread. It ignores the parietal pelvic glands, the presacral, pre-aortic, and para-aortic glands and tissue. Spread occurs to these zones in highly malignant growths, very advanced growths, and in young subjects.

The extended combined excision involves mobilization of the splenic flexure, ligating the inferior mesenteric artery flush with the aorta and the inferior mesenteric vein as it passes behind the pancreas. The cellular tissue is cleared from in front of the aorta and vena cava and the left para-aortic glands are removed. The dissection is carried along both iliac vessels, removing the internal iliac and obturator groups of glands.

The added risk of the operation lies in the blood supply to the colostomy which is now derived from the left branch of the middle colic artery. This is usually adequate except in obese subjects or in the presence of arterial disease (atherosclerosis). The increased operating time required to complete the extensive dissection also adds to the risk of this operation

Excision of the Rectum with Sphincter Conservation

(1) Anterior resection.

(2) Abdomino-anal excision.

It is possible to remove a rectal cancer radically by these operations in 25 per cent of rectal growths.

Recurrence at the suture line can be eliminated if, after the rectum is clamped at least 5 cm. below the lower edge of the tumour, the rectal stump is meticulously irrigated and cleaned with 1:500 perchloride of mercury. THIS IS THE KEY STEP OF THE OPERATION.

There is no need to limit in any way the removal of the upward zones of lymphatic spread. In fact, for anterior resection with mobilization of the splenic flexure there is enough viable bowel in most cases to allow the inferior mesenteric artery to be tied at its origin. The inferior hæmorrhoidal arteries are adequate supply for the rectal stump after division of the lateral ligaments and mid-hæmorrhoidal vessels. There need be no limitation of the removal of the lateral zones of spread.

The operations are more difficult than a combined excision, they carry a higher morbidity rate, and at times need to be staged

In order to eliminate the possibility of growths in the lower sigmoid colon being included in series of rectal carcinomas removed by conservative excision it is essential that the following data should be recorded accurately:

(1) The distance of the lower edge of the growth from the anal margin as measured through the sigmoidoscope.

(2) The relationship of the growth to the peritoneal reflection.

(3) The relationship of the tumour to the sacral promontory.

(4) Measurements obtained on the excised specimen after fixation.

(5) The distance of the anastomotic line from the anal margin assessed at examination in the follow-up period.

The majority of anterior resections can be managed as one stage operations.

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Complications before operation

The age group in which the operation is commonly practised brings with it the problems of old age, heart, lung, and kidney disease. References have already been made to obesity and weight reduction.

Constant bleeding of the growth and the specific effect in many cases produces a secondary anæmia. Steps must be taken to correct this anæmia and to provide an adequate supply of blood for administration during and after operation.

Intestinal obstruction, usually chronic but occasionally subacute, may have to be corrected, before the primary tumour is resected, by a preliminary colostomy. Rarely growths at and above the peritoneal reflection may perforate and present as a spreading (fæcal) peritonitis.

Complications during operation

(1) Opening of the rectum and local soiling, usually during the anterior perineal dissection.

(2) Injuries to the ureters and bladder (Graham and Goligher (1954)).

(3) Damage to veins and arteries especially on the pelvic walls, resulting in primary arterial and venous hæmorrhage.

Complications after operation

Immediate. (1) Shock. In an expeditiously performed excision without blood loss shock is slight Hæmorrhage during the operation, although replaceable, adds to the degree of shock.

(2) Hæmorrhage (reactionary) may occur from arteries and veins in the pelvic wall and a constant watch must be kept on the perineal dressing in the immediate post-operative course.

Hæmorrhage has also been known to occur from a slipped pedicle ligature or round the colostomy.

Secondary hæmorrhage in infected perineal wounds may occur later in the post-operative period.

(3) Retraction of the colostomy due to insecure fixation or gangrene from inadequate blood supply has occurred.

(4) Ileus. There is always a degree of ileus which lasts a few days. The oral intake of solids and fluids is forbidden for 48 hours at least and the body fluid and mineral intake is maintained by intravenous therapy.

When ileus persists, the question of intestinal obstruction and peritonitis arises and the distinguishing between ileus and mechanical intestinal obstruction presents great difficulties

Peptic ulceration with perforation or hæmorrhage is a not uncommon complication which gets under a surgeon's guard. The careful taking of pre-operative history may alert staff for such a possibility There is no doubt that the anxiety of a major operation for so serious a disease increases ulcer activity.

(5) Urinary retention and infection. In an operation which involves the base of the bladder and the pelvic parasympathetic and sympathetic nerves it is not surprising that urinary function is disturbed After operation the bladder may be drained by a urethral catheter or a small suprapubic catheter (Riches) in males Females should be

catheterized eight-hourly. Sometimes urinary function returns in 24 hours and when it does artificial aids for draining the bladder may be discontinued. On an average, however, their help will be needed for at least four days. In nervous patients and where the operation has been very extensive, a state of urinary retention persists for periods up to 14 days. When retention persists beyond this time serious damage to the nervi erigentes is probable. In these cases the retention may persist for months and function may only return with incontinence. The question of transurethral resection of the bladder neck should be considered in conjunction with a urologist.

Mild degrees of urinary infection are common, especially if retention of urine has occurred. The organisms should be tested for antibiotic sensitivity and the appropriate drugs administered.

(6) Peritonitis. Twenty years ago this was still a common occurrence. Today it is almost unknown. Antibiotics, pre-operative preparation, and avoidance of peritoneal contamination at operation have all contributed to its eradication.

(7) The aggravation of heart and lung disease will have to be treated and in some cases post-operative confusion and mania may result in the loss of the co-operation of the patient.

Later Complications

(1) Intestinal obstruction. There is a high incidence of intestinal obstruction from: prolapse of knuckles of small intestine through the sutured pelvic peritoneum, adhesions to the ligature pedicle, and strangulation of intestine through the lateral space formed by a colostomy. This subject has been discussed by Goligher.

(2) Colostomy stenosis may occur, resulting in irregular colostomy actions. It is due to constriction of the cut end of the circular muscle constricting the opening at the skin edge. It may be avoided if a trephine colostomy with primary epithelial apposition is practised

(3) Hernia may occur through the long abdominal incision, round the colostomy, and through the perineal wound (sacral hernia) and bring with it the complications associated with hernia.

Complications Peculiar to Anterior Resections. These are complications which occur at an anastomosis and consist of small areas of breakdown of the anastomosis to complete disruption

The first sign that the anastomosis is not healing perfectly is the passage of flatus or liquid fæcal material through the extraperitoneal drain and the formation of an abdominal fæcal fistula. In most cases this is minimal and of a short duration.

Where more extensive breakdown has occurred a right upper transverse colostomy must be performed, if this has not already been done.

Pelvic Abscesses. As a result of breakdown of the anastomosis pelvic abscesses may form. These can be drained through the suture line into the rectal lumen.

Treatment of Advanced and Incurable Rectal Cancer

By direct extension a rectal growth may involve the bladder, urethra and prostate or the vagina, cervix, and uterus.

In the female, the additional removal of the reproductive organs by a Wertheim hysterectomy presents no exceptional difficulties.

In the male, only a pelvic clearance can ensure the removal of growths invading the urinary organs. Careful consideration must be given to every aspect of the case before embarking on these procedures. The steps of the operation may be summarized as follows:

(1) The inferior mesenteric artery, middle sacral, and internal iliac arteries are first ligated. The excision of the pelvic viscera can then be effected with reduced blood loss.

(2) No attempt is made to re-form the peritoneal floor.

(3) The ureters are anastomosed to a short isolated ileal segment and exteriorized through the right abdomen as a urinary ileostomy.

(4) A terminal left iliac colostomy is established.

(5) The perineal wound is carefully and completely sutured and the intestines allowed to fall into the pelvic basin.

If widespread irremovable secondaries are present it will still in many cases help the patient if the rectum and primary growth are removed. Occasionally liver destruction is so great and the expectation of life so short that in these cases cutting away the primary tumour with a diathermy loop through an operating sigmoidoscope affords a measure of palliation

A colostomy alone should be avoided at all costs and only used if other methods have failed to relieve distressing intestinal obstruction.

The position today of a patient suffering from a carcinoma of the rectum can be summarized as follows· The primary tumour can be resected or excised (resectability rate) with benefit in at least 90 per cent and probably in 95 per cent of cases. With such a high resectability rate the average mortality rate is 10 per cent. Many individual surgeons, however, have reduced their personal mortality rate to between 2 and 5 per cent.

In 15 per cent of cases, where the primary tumour is removed, the disease has become widespread and can no longer be eradicated by surgery; the operation is therefore palliative and may be judged to be so from:

(1) Incomplete removal of extensive local spread.

(2) Involvement of inaccessible lymphatic glands.

(3) Widespread peritoneal involvement

(4) Spread to distant organs such as lungs or liver.

Hope need not be abandoned for all these palliative cases. Liver and lung resections are now successfully performed for secondary malignant deposits. The steady improvement in radiotherapy demands a constant review of the possibilities of pre- and post-operative irradiation and intraperitoneal radio active isotopes may one day be of assistance for intra-peritoneal growth.

If only one case can be salvaged from a large number in this group the effort for many has been worthwhile.

In cases where the surgeon has hoped to eradicate the disease the five year cure now stands between 45 and 50 per cent.

A surgeon's efforts today should therefore be aimed at removing the primary tumour in 90 per cent of cases, with a mortality rate not above 5 per cent and a 50 per cent five year cure, and at achieving a degree of palliation for incurable cases.

Reference

Lloyd-Davies, O. V. (1939) *Lancet*, 2, 74.

CONNECTIVE TISSUE TUMOURS

SECONDARY CARCINOMA OF THE RECTUM. The rectum can be involved by direct spread from growths arising in neighbouring viscera, the two commonest examples being carcinoma of the prostate and cervix uteri.

Carcinoma of the prostate may spread round the rectum to form a stricture and the difficulty of diagnosing between a rectal stricture secondary to carcinoma of the prostate and an atypical, stricture-forming, anaplastic primary rectal growth is not wholly clinical. The difficulty is shared by the pathologist with the biopsy slide under the microscope.

Carcinoma of the cervix involves the rectum. The opportunity occasionally presents, before the bladder and uterus are involved, for a Wertheim hysterectomy combined with a radical abdomino-perineal excision of the rectum. Pelvic clearance, Brunschwig (1948) is the only alternative in more advanced growths. The ureters are led into a short ileal conduit and a urinary ileostomy and permanent colostomy are established.

Carcinoma of the ovary also invades and erupts into the rectum before a massive pelvic tumour has developed and may explain puzzling histology shown at biopsy.

Tumour metastases in the pelvic peritoneal pouch can involve the rectum. Secondary carcinoma of the breast has been reported as invading the rectum in this way.

Lastly, adenocarcinoma tumour fragments can implant on granulation tissue, and there are known examples of carcinoma of the colon implanting on simple chronic anal lesions such as fistulæ and fissures. A patient with a long standing anal lesion that has undergone a malignant change should be examined carefully for a growth higher up in the colon. A carcinoma of the sigmoid colon can prolapse into the recto-vesical and recto-uterine pouch, and become adherent to and erupt into the upper part of the rectum.

A rectal growth then can be secondary to a colon growth by direct or indirect spread.

Leiomyoma. Tumours of unstriped muscle occasionally arise in the rectum. Their mobility and a benign report on the biopsy often lead up to their removal by enucleation which can be accomplished with reassuring ease.

Recurrence within six months following local operations is so frequent that leiomyomatous tumours should be regarded as malignant. It is almost impossible to differentiate from a biopsy specimen between a benign leiomyoma and its sarcomatous form.

Hæmangiomas may occur as localized lesions or diffuse lesions involving the whole of the rectum and pelvic colon. Small localized lesions may be responsible for unexplained rectal bleeding. The diffuse cavernous angiomata present as massive recurrent hæmorrhages. At sigmoidoscopy large pulsating vessels can be seen in the submucosa. It is usually necessary to excise the rectum and pelvic colon; but Gabriel (1948) describes a case treated in stages by colostomy, ligature of the inferior mesenteric artery at the aortic bifurcation, and then later colostomy closure

Sarcomata of rectum may occur as a lymphosarcoma, reticulosarcoma, spindle celled sarcoma or leiomyosarcoma.

Lymphosarcoma is the commonest type and may present as a localized tumour or a diffuse nodular infiltration of the rectum.

Sarcomata may be recognized clinically as a rounded tumour projecting far into the lumen of the bowel, being wholly or partly covered by rectal mucosa. Their bad reputation as rapidly fatal growths is probably due to the difficulty in making a histological distinction between sarcoma and anaplastic carcinoma.

Dukes (1947) has reported three cases of lymphosarcoma surviving 5 years, 4 years, and 3 years after radical excision. Radical excision should be combined with radiotherapy and prognosis is not as hopeless as was previously believed.

Lymphomata. These are uncommon sessile or pedunculated tumours arising in the anal canal. They may be multiple. In structure they resemble lymphatic tissue and are benign.

Miscellaneous Tumours

(1) Carcinoid Tumours (Argentaffinomata) arise along the whole length of the intestinal tract with the highest incidence in the appendix and the lower ileum. They arise from specialized cells in the bases of the crypts of Lieberkühn. The cells, named after Kultschitzky who first described them, have an affinity for silver stains. The cut surface of the tumour is characteristically yellow or orange. The tumour is slow growing and of low grade of malignancy.

Symptoms and Signs. It is unusual for carcinoid tumours of the rectum to give rise to symptoms. They are discovered during a routine rectal examination and present as a polyp, a small sessile nodule, submucous plaque or induration. Raven (1950) has given a full description of these tumours.

Treatment. A small tumour should be treated by local excision; recurrence is exceptional. If there is a recurrence after local excision, or with large tumours and surrounding infiltration, a radical excision must be performed.

(2) Malignant Melanomata usually occur at the anal verge or in the anal canal. They are highly malignant and even early cases treated by the most radical excision rapidly succumb to distant metastases.

They present in two forms: sessile or polypoid (Raven, 1948) and have a characteristic dark brown purple colour not unlike a thrombosed pile or anal hæmatoma. Metastases are characteristically black. Peritoneoscopy is indicated before undertaking a radical excision, hepatic metastases can be easily seen and, when present, radical surgery is contra-indicated owing to the short expectation of life.

References

Dukes, C. E (1947) *Brit. J Cancer*, 1, 30 (sarcomata)
Brunschwig (1948) *Cancer*, 1, 177.
Raven, R. W (1950) *Proc. Roy. Soc Med.* 48, 675.
Raven, R. W. (1948) *Proc. Roy. Soc. Med.* 41, 469.

PROLAPSE OF THE RECTUM

Prolapse of the rectum is a protrusion of the rectal wall through the anal orifice. If only the mucous membrane protrudes, it is known as a partial prolapse, but when all coats of the rectum are involved it is known as a complete prolapse. It occurs in infants and the aged, in males and females, but is six times more common in females than in males.

The problems of rectal prolapse are so different in children and in adults that it is proposed to discuss the condition in these two groups.

Rectal Prolapse in Children

It occurs from the age of nine months to three years and is undoubtedly associated with the passage of a formed stool.

It can occur in an ill or a healthy child. The illnesses which predispose to the condition are:

(1) Wasting disease which results in the loss of supporting fatty tissue round the rectum.

(2) Diseases causing a repeated and sustained raising of the intra-abdominal pressure, such as whooping cough, bronchitis, and asthma.

(3) "Diarrhœa and vomiting" which not only results in loss of supporting fatty tissue and raising of the intra-abdominal pressure but causes a loosening of the rectal attachments by frequently repeated eversion of the anus at defæcation.

(4) By the abnormally frequent contractions and relaxations of the sphincter muscles caused by the irritation of threadworms.

(5) Neurological conditions such as spina bifida and meningocœle which result in paralysis of the pelvic floor muscles.

(6) Mental deficiency where co-operation in bowel training is difficult to achieve.

With the present standards of child welfare many of the diseases associated with rectal prolapse, such as marasmus and rickets, are now extremely rare and the problem which confronts the surgeon today is that of rectal prolapse in a healthy child.

Diagnosis. The mother will usually be the first to notice a mauve, congested swelling at the anus when she goes to cleanse the child after defæcation. Her attention may be drawn to the fact that something is wrong by spots of blood in the chamber. The condition is quite painless The difficulties in diagnosis lie in the fact that at the time of consultation the prolapse is not evident, neither will the child produce it on request. It is also remarkable that if the child is admitted to hospital for observation the prolapse does not appear and is rarely seen.

There is no doubt that the spoilt child with over anxious parents comes to regard the prolapse of his rectum as an accomplishment which will immediately focus a great deal of fussing and attention on himself. The substitution of familiar home surroundings and irresolute parents by the discipline of a hospital ward and its sister is frequently sufficient to prompt a child to keep his rectum in place.

The main factor responsible for rectal prolapse in otherwise healthy children is straining on an empty rectum. This may be due to over anxiety of the parent for the child to achieve a daily evacuation, or neglect in training a child in the after breakfast habit and allowing him to evacuate the intestinal residue at irregular times after the rectum has become overloaded. In the former case, the trial of defæcation is transformed into a prolonged affair when the child is encouraged to defæcate whether there is a physiological need or not, his attention to the call of nature being frequently distracted by toys and his mother's attempts to entertain him.

Treatment. It should be emphasized that there is a strong tendency for spontaneous cure without any elaborate treatment. If there is an underlying factor it must be recognized. Those in charge of the child should be instructed in the mechanism of the gastro-colic reflex and the desirability of establishing an after breakfast habit. If necessary, a glycerine suppository may be inserted into the rectum before breakfast; by

the time breakfast is over this will have added its effect to the normal stimulus to defæcate and a successful evacuation will result.

Care should be taken that the child is sitting on a correctly shaped chamber. Some of these utensils are so designed that undue traction is placed on the skin of the buttocks and perineum, pulling apart the rectal sphincter and weakening its hold on the rectum.

Later on, when a prolapse has become established, a period of defæcation in the lying down position may be necessary. It is usually advisable to admit the child to hospital

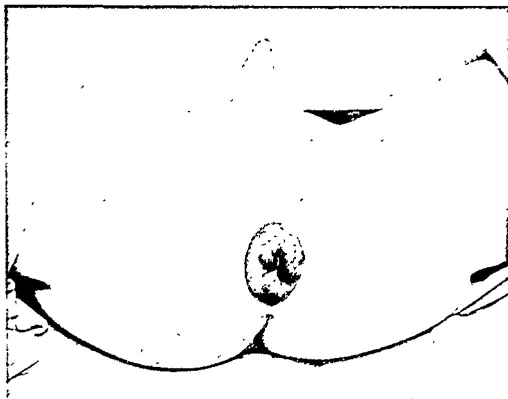


Fig. 231. Rectal prolapse in a male child.

as mothers of these children seem unable to discipline themselves or their children to do this. At the commencement, a small soap and water enema is given in the left lateral position and the child soon learns to evacuate the enema lying on his side. Subsequently he learns to defæcate in this position if encouraged to do so at the same time each day and in many cases this may effect a cure.

Submucous Injections. The injection of 5 per cent Phenol in almond oil into the submucosa of the rectum produces a reaction which fixes the mucosa of a partial prolapse and will certainly help in a complete prolapse. The injection should be given under local anæsthesia and if the rectum can be prolapsed, a ring of solution is injected round the apex of the prolapse Further areas may be injected at a lower level according to the size of the prolapse. If the rectum cannot be prolapsed the injections are given through a proctoscope.

Perirectal Injections. Various solutions have been used to inject the perirectal tissues with the object of causing a fibrosis and fixing the rectum to the pelvic walls. Absolute

alcohol 1·5 ml. injected into the perirectal tissues on the postero-lateral aspects has given good results. Double the quantity of 5 per cent Phenol in almond oil may be used similarly.

Perianal Suture. A circular suture of No. 1 chromic catgut may be inserted round the perianal space to constrict the anus to a size which will allow a soft stool to pass but will not allow the rectum to prolapse. This is a very reliable surgical procedure in stubborn cases.

Methods of treatment resulting in post-operative pain or discomfort, especially those where alcohol is injected under the anal mucosa, transform prolapsing of the rectum from a painless into a painful event. There is no doubt that a child quickly appreciates the change and contracts his sphincter muscles to prevent the prolapse occurring, thus assisting in his own cure.

The time-honoured method of strapping the buttocks together has no application in the usual case where the prolapse only occurs on defæcation. If, however, as in mental defectives, the prolapse is constantly down, strapping the buttocks together may assist in keeping the prolapse in place during the day.

It is important that the child's parents be instructed to reduce the prolapse correctly. Firm, sustained pressure through a pad of damp cotton wool will lead to rapid reduction. If difficulty is experienced in reduction the child may be lifted up by his legs with his head hanging down; the intestines will fall into the upper abdomen and traction will be exerted on the prolapsed rectum from within.

The author has never seen an irreducible or strangulated prolapse in a child and if this should occur then presumably more serious surgical methods may be required to deal with the condition.

Rectal Prolapse in Adults

Partial Prolapse

In partial prolapse of the rectum a double layer of mucous membrane protrudes from the anus The protrusion is rarely more than $1\frac{1}{2}$ in. long and may be confined to the right or left halves of the rectum and anus or may involve the whole circumference of the bowel lumen By grasping the prolapse between the thumb and forefinger it is easy to appreciate that the prolapse consists only of mucous membrane.

Ætiology. The commonest cause of partial prolapse in adults is confluence of the mucous membrane of prolapsing hæmorrhoids. On the right side, the right posterior and right anterior hæmorrhoids combine to form a right lateral partial prolapse; on the left side, the left lateral hæmorrhoid combines with its two secondary satellites to form a left lateral partial prolapse.

It is a frequent sequel to operations on extensive fistulæ with high internal openings where a major part of the sphincter muscle has to be divided. It often follows the Whitehead type of operation for hæmorrhoids.

Straining at defæcation due to chronic constipation, either primary or secondary, is a contributory factor. Efforts to overcome urinary obstruction, especially prostatic enlargement and urethral stricture in the male, have the same effect.

In females, damage to the perineum and rectal sphincters at childbirth are undoubtedly contributory factors. A definite group of cases occur in elderly people who are undergoing a general tissue atrophy and loss of muscle tone. Clinically, these

patients are characterized by an almost complete absence of fat from the ischio-rectal fossa and lax, atonic sphincters.

In the tropics it is not uncommon for the rectal mucous membrane to prolapse during the course of an amœbic dysentery. Failure to recognize the underlying pathology may result in disaster if attempts are made to excise the prolapsing tissue. Adequate antiamœbic treatment will lead to a spontaneous cure.

Symptoms. The symptoms often follow on, and are indistinguishable from, those of the hæmorrhoids which precede them. At first the mucosa prolapses only on defæcation and is associated with bleeding and rectal discharge. Later on, the mucosa is permanently prolapsed, the perianal tissues are constantly moist and pruritus ani plagues the patient.

Pathology. The prolapsed mucous membrane becomes congested and inflamed, its pale salmon colour changes to a bright shiny red. In long standing cases, squamous *metaplasia spreads over the mucous membrane, either in continuity with the skin of the* anal canal or in irregular patches.

Treatment. Before deciding on any course of treatment a careful search to eliminate and, if present, treat any underlying factor must be made. It is not out of place to repeat that a complete digital, proctoscopic, and sigmoidoscopic examination should be made. Constipation, due to the obstruction of a rectal growth, may result in the straining down of rectal mucosa.

Submucous Injections. Injection of the submucosa with 5 per cent of Phenol in almond oil with 2 gr. of Menthol to the ounce can give a great measure of palliation and sometimes may effect a cure. This method of treatment is especially indicated in the old age group where attempts at excision are almost invariably followed by recurrence. The technique is the same as for the injection of hæmorrhoids except that larger quantities of solution will have to be injected and a greater number of injections will need to be given.

Ligature and Excision. Where the partial prolapse is secondary to hæmorrhoids it should be treated as such The prolapsing mucosa is grouped and ligated at the sites of the three primary hæmorrhoids. Its excision leaves three interhæmorrhoidal strips of anal mucosa 0.5 cm. wide. At the finish of the operation these strips of tissue may appear loose and untidy but they almost invariably shrink down. If skin tags develop they can be dealt with at a second operation under local anæsthesia and the patient should be warned before operation that this might be necessary.

Goodsall's Ligature. This is particularly applicable when the partial prolapse is unilateral. Two curved round-bodied needles are threaded one third and two thirds *down a long ligature of No. 3 chromic catgut The prolapsing mucous membrane is* transfixed so as to divide it into three equal parts, the catgut is divided at the needle eyes, and the corresponding ends are tightly tied. Redundant mucosa is then excised. (Fig. 232.)

Complete Prolapse

It is unfortunately not known how many cases of complete prolapse in childhood persist into adult life and, for obvious reasons, it is only rarely that adult patients with complete rectal prolapse can give a history from infancy.

Age and Sex Incidence. It is six times more common in women than in men. Although the ratio of married to single women over the age of 15 is 3:1, the ratio of married to

single women suffering from rectal prolapse is 3:2. There is, therefore, a relatively high incidence of rectal prolapse in single women.

The condition may appear at any age, in males the highest age incidence is in the second and third decades and in females the maximum incidence is in the sixth decade. There is, however, a corresponding incidence peak in the second and third decades in females and in both sexes there is a small incidence peak in septuagenarians and octogenarians.

Ætiology. The ætiology is best understood by firstly considering what keeps the rectum in place. The rectum is suspended by its continuity with the pelvic colon. If the colon and its mesentery are short it is unlikely that the rectum will prolapse. It is kept in place by various fascial planes and especially the strong lateral ligaments which pass

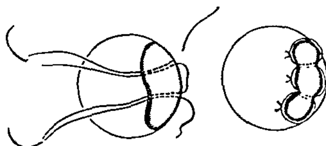


FIG 232. Diagram illustrating Goodsall's ligature with two needles.
(From "British Surgical Practice," Vol VII, Butterworth's, London)

from the sides of the rectal ampulla to the lateral pelvic walls. The levator ani and the sphincter ani muscles also play a very important part in keeping the rectum in place. The various peritoneal ligaments between the rectum, sacrum, bladder, and vagina make a small contribution. The rectum is therefore suspended from above, fixed at the sides, and held up from below. Anything weakening one or more of these mechanisms may lead to a complete rectal prolapse.

Next, it is important to realize how a complete rectal prolapse develops. The starting point of the prolapse is an invagination of the anterior rectal wall into itself at the peritoneal reflection, just above the levator ani muscle. As the invagination enlarges, it drags down the lateral and posterior walls. In other words, the rectum prolapses through a weak spot in the musculo-fascial pelvic diaphragm and the condition is comparable to a sliding hernia (Moschcowitz, 1912) For a hernia to develop, two factors must be present. There must be a weak spot in the abdominal cavity and increased intra-abdominal pressure, and there is no doubt that both these factors are to be found in this condition. The weak spot is the exit of the rectum through the pelvic diaphragm In lesions of the spinal cord or cauda equina (Butler, 1954), resulting in paralysis of the muscles of the pelvic floor, rectal prolapse, frequently associated with urinary incontinence, may result. It is seen also in cases of spina bifida. In cases of anal incontinence due to complete division of the ano-rectal ring at fistula operations the support of the rectal sphincter is lost and a complete prolapse may result. Increased intra-abdominal pressure may be due to the straining of chronic cough, constipation, and urethral obstruction There is no doubt that the deposition of fat in the omentum and abdominal viscera (especially in the pelvic colon and its appendices epiploicæ) can add its weight to pushing the rectum on its way outside.

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Symptoms. The patient may be aware at first of a discharge of mucus. This may l followed by protrusion of the bowel on defæcation. Later, the prolapse descends on tl slightest exertion. The prolapse stretches and weakens the sphincter and the patient final becomes incontinent and is confined to the house. It is worth commenting that patien will remain silent about, and bear with, the condition for a long period before seekir and accepting professional advice.

Signs. The rectum protrudes for a distance of 4–6 in. It consists of two thickness of complete bowel wall and between the two layers anteriorly is a peritoneal pouch in



FIG 234 Complete rectal prolapse in a male aged 19.

which abdominal contents may descend. The mucosa is arranged in a series of thi circular folds and the anterior wall of the prolapse is longer than the posterior wa This gives the prolapse a curve with a concavity backwards There is frequently a chror ulcer situated at the apex and sometimes ulceration may occur at its base. When t prolapse has been reduced the resting tone of the sphincter muscles is found to almost negligible. Light traction on the anal margin causes the anus to open a remain patulous (Fig. 235). After passing the finger into the rectum and requesting t patient to pull in and contract his sphincter muscles, he will often do exactly the opposi i.e. bear down and relax. There seems to be a complete confusion of muscular mecha ism. The important point, that it is through the anterior rectal wall that the prolap starts, can be demonstrated by the fact that pressure on the perineum in front w effectively prevent the prolapse coming down, in spite of the patient straining. Simil pressure posteriorly will have no restraining effect. After inserting the finger into t rectum and asking the patient to strain, the anterior part of the rectum will be noted descend first.

Injury to the perineum and sphincter muscles at parturition is undoubtedly contributory but parturition is not the important factor that it might at first seem to be. It *remains a puzzle why healthy young men and women in the second and third decades* should suddenly develop a rectal prolapse with a weak, atonic sphincter. There are many speculative possibilities, such as localized poliomyelitis affecting the anterior horn



Fig 233 Complete rectal prolapse in a female aged 21

cells supplying the pelvic muscle, or some form of venereal infection which is not as yet recognized. Lastly, a complete rectal prolapse may be pulled down by a rectal tumour such as a villous papilloma, a large adenoma, or a small carcinoma. The rectum interprets the tumour as a fæcal bolus and so successful are its efforts to extrude that the whole rectum finally prolapses.

Mental Deficiency. Complete rectal prolapse is undoubtedly associated with mental deficiency. In mental institutions there is usually to be found an inmate with a complete rectal prolapse. Grown up patients with rectal prolapse often have the mentality of a young child. Apparently normal people after operation for a rectal prolapse may become mentally confused, delusional, and sometimes maniacal

constricted, the prolapse becomes strangulated and gangrenous and it is interesting to recall that the first amputation of a prolapse was performed for this complication.

(5) Perforation of the Anterior Rectal Wall. Cases have been reported where the anterior rectal wall has perforated into its underlying hernial sac of peritoneum and the abdominal contents have cascaded out between the patient's legs.



Fig 236 Inflamed, ulcerated prolapse in a male of 34

Treatment. The difficulties of treatment arise from the widely varying types of patient presenting for treatment and in the choice and application of method. It is hardly to be expected that a single surgical panacea exists to cure a complete rectal prolapse in a healthy young adult of 25, an obese multiparous woman of 60, or a frail, senile octogenarian

It sometimes happens that a complete rectal prolapse is first recognized when traction is applied to the pile pedicle preliminary to hæmorrhoidectomy. If this occurs, it is better to refrain from an unconsidered, improvised operation and reconsider the case *in all its* aspects for operation at a later date. The importance of assessing a patient from the mental aspect before operation cannot be too strongly stressed. It may forewarn the nursing staff of post-operative complications, of delusions, hallucinations, or even mania.

Differential Diagnosis

Complete prolapse must be distinguished from partial prolapse and not infrequently a prolapsed rectal or colonic tumour is mistaken by the patient or his doctor for a rectal prolapse.

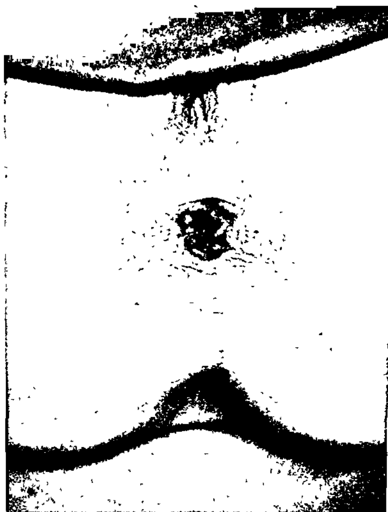


Fig 235 Patulous anus

Complications

(1) Uterine and Vaginal Prolapse Complete prolapse of the uterus may complicate a rectal prolapse, and as a rectal prolapse is really a disease of the pelvic floor it is not surprising that this should happen (Fig 237).

(2) Inflammation In long standing cases a proctitis develops, the mucosa loses its circular folds and becomes smooth, bright red, and glistening with excess of mucus secretion (Fig. 236)

(3) Irreducibility. Owing to œdema from inflammation a rectal prolapse may become irreducible, but in spite of its irreducibility patients sometimes continue to neglect the condition.

(4) Strangulation. Sometimes the blood supply to a rectal prolapse becomes

but the fact that patients return to have this done is a testimony to the method. It may also supplement other forms of treatment. Mention has already been made of its catgut modification as applied to children.

(2) *Repair of the levator ani Muscle.* This may be done either from above or below. The levator ani muscle itself may be wasted and atrophic, especially in cases



Fig 238 Recurrent rectal prolapse in a woman of 65 following recto-sigmoidectomies

where its nerve supply is injured and in these cases the only value of attempting this procedure would be to give the additional support of its sutured fascial covering.

Suture of the levator ani muscle in front of the vagina is possible from above and forms an integral part of Roscoe Graham's operation (1942). The levator ani may also be repaired from below as in a perineorrhaphy (McCann, 1928) and the suturing of the levator ani has been recommended as an additional step in Miles' operation of recto-sigmoidectomy (Cohn, 1942)

(3) Attempts to strengthen the areolar tissue attachments are to be found in the various rectopexies and sigmoidopexies that have been devised and practised from time

Consideration of the surgical anatomy of a complete rectal prolapse gives the clues to the structures to which therapy should be directed—

(1) The sphincter ani muscle is stretched and atonic.

(2) The gap in the levator ani muscle, through which the rectum passes, is sometimes so large that an adult fist can be pushed through with ease.

(3) All the connective tissue attachments of the rectum are stretched and loosened. The lateral ligaments, instead of being strong, thick structures are long, thin, and attenuated.

(4) The recto-vesical or recto-uterine pouch is seen as a large deep basin. The peritoneum covers the posterior vaginal wall; the recto-vaginal septum is practically



(From "British Surgical Practice," Vol II, Butterworth's, London)
FIG 237 Complete uterine and rectal prolapse

non-existent. The whole of the rectum above the levator ani is invested with peritoneum which also lines the upper surface of the levator ani muscle. The rectum can be easily lifted up from the sacrum and coccyx and the mesorectum itself is a long pliable structure.

There are in existence some forty different surgical procedures which have been employed to treat a rectal prolapse, all of them designed to correct one or more of the above abnormalities.

(1) *To correct a Lax, Atonic Sphincter* It is well recognized that both pre-operatively and post-operatively the patient must be instructed in, and practise, sphincter ani muscle exercises. This can be supplemented with surging Faradism

Attempts to narrow the sphincter by plicating it meet with little success The stretched, dilated external sphincter available for suturing from below does not take kindly to reconstruction.

Thiersch's operation of inserting a silver wire in the perianal tissues and thus narrowing the anal orifice is of benefit in many cases The wire may break and require re-insertion

Proctitis is also seen in disorders of bowel function where the patient retains large scybalous masses in the rectum and makes habitual use of purgatives and enemata to relieve the disorder.

Proctitis can also be a complication of venereal diseases, especially lymphogranuloma venereum and gonorrhœa. In gonorrhœa the disease may be a primary rectal infection caused by rectal coitus and homosexual practices or secondary to the usual urogenital involvement. The condition is relatively symptomless and usually occurs in a sub-acute form. Anal irritation, discharge of mucopus and bleeding, with small abscesses at the anal margin suggest the diagnosis. The diagnosis depends on the identification of gonococci in smears of mucopus.

Proctitis may develop as a complication of radium treatment of carcinoma of the cervix and follows X-ray radiation of the pelvis. The proctitis starts as a rule at the end of the first week of treatment and is heralded by a watery blood-stained discharge and tenesmus.

Non-Specific Proctitis and Procto-Colitis

The commonest form of proctitis in this country is the type of proctitis which is known as hæmorrhagic proctitis and is closely allied to, or part of, the group of diseases included in the term "ulcerative colitis." The patient presents complaining of rectal bleeding on defæcation and usually describes his complaint as "piles." Examination, however, shows that there are no hæmorrhoids but the rectum itself is hyperæmic and bleeds easily to swab touch, producing small petechiæ. Mucopus is present in the lumen. Sometimes the condition is confined to the lower 6 cm. of the rectum and anal canal, the bowel above this level being apparently completely normal. No local application has any effect on the condition, except to make it worse, and nothing taken by mouth or parentally is known to have any lasting effect. The disease can remain localized for 10 years and then suddenly develops into a diffuse inflammation of the whole of the rectum and colon. When a longer segment of bowel is involved, the condition is known as "procto-colitis." All that can be done is to reassure the patient and keep a regular check on the hæmoglobin level and, if a secondary anæmia is developing, to give the necessary drugs or blood transfusions to correct it.

RECTAL STRICTURE

Reference has already been made to the various types of malignant stricture of the rectum (page 496, Differential Diagnosis, No. 10). A malignant stricture may be produced by a primary rectal tumour or by the secondary invasion of growths in related viscera such as the cervix uteri and prostate. Malignant deposits in the recto-vesical and recto-uterine pouches (secondary to carcinoma of the stomach, colon, or breast) can cause a rectal stricture by direct invasion or from a secondary fibrous tissue reaction. It is thus necessary to point out that the differential diagnosis between a malignant and a non-malignant stricture is sometimes extremely difficult, especially when the rectal growth presents in atypical forms such as the diffuse tubular stricture which has been called a "cylindroma."

Prostatic carcinomas can also stimulate a fibrous reaction round the rectum which may give rise to a rectal stricture. The diagnosis of a tubular type of stricture recently

to time. Some of them aim at producing perirectal adhesions by aseptic inflammation. Many of them are at fault, being directed to the posterior wall of the rectum when it is the anterior wall which prolapses first and starts the trouble. The author has dissected out and isolated the lateral ligaments, shortened them by plication and resutured them at a higher level to the postero-lateral pelvic periostium and believes this to be a sound procedure.

(4) Obliteration or removal of the redundant peritoneum lining the pelvic basin is practised in Moschcowitz's and Roscoe Graham's operations. Resection of the rectum from above (anterior resection) or from below (rectosigmoidectomy) removes the long mesorectum and redundant bowel and there is no doubt that a formal anterior resection fixes the rectum very firmly in place (Muir, 1955). Rectosigmoidectomy, which was popularized by Miles (1933) has been the method of choice for the last twenty years. It is a safe operation without operative mortality and, if properly performed, with few complications. Careful analysis of the results of this operation, however, has shown that there is at least a 60 per cent recurrence rate and in those cases where there has been no recurrence, at least 50 per cent are incontinent. On the other hand, the operation can be brilliantly successful in young subjects before the sphincter mechanism has deteriorated. It has an obvious application when the prolapse has become irreducible or strangulated (Fig. 238).

There are cases where every form of treatment seems to fail. They are usually stout, obese women with a sphincter weakness or injury amounting to complete paralysis. In these cases the question of an excision of the rectum and a permanent colostomy should be seriously considered. With a well-constructed colostomy the patient can lead a normal life in contrast to the discomfort and confinement that a large rectal prolapse imposes.

Summary. If, as Moschcowitz suggested, complete prolapse of the rectum is regarded as a sliding hernia of the rectum, then the principles of hernia surgery must apply to its treatment. The three principles in repair of a hernia are. to remove the hernial sac, to repair the internal ring, and to narrow the external ring. Applied to a rectal prolapse, this may be interpreted as removing or obliterating the pelvic peritoneum, repairing the exit of the rectum through the levator ani muscle (internal ring) and narrowing the anal orifice (external ring) It is unlikely that all three principles can be applied in every case but one or a combination may give a degree of palliation for which a patient may indeed be grateful

References

Moschcowitz, A. V. (1912) *Surg Gynec Obstet.* **15**, 7.
Butler, E C. B (1954) *Proc. Roy Soc Med* **47**, 521.
Graham, R. R (1942) *Ann Surg* **115**, 1007.
McCann, F. J (1928) *Lancet*, **1**, 1072.
Miles, W. E. (1933) *Proc. Roy. Soc Med* **26**, 1445.
Muir, E. G. (1955) *Proc Roy. Soc. Med.* **48**, 43

PROCTITIS

The rectum may be inflamed as a part of a generalized specific disease of the large intestine as in bacillary dysentery, amœbic dysentery, or Bilharzial infections of the rectum and colon.

proctocolectomy and a permanent ileostomy. A rectal stricture is a contra-indication to any conservative operation in ulcerative colitis.

Lymphogranuloma Venereum is only rarely seen in this country and then it tends to be localized at large ports with a shifting, seafaring population. The development of the disease seems to be connected with climatic conditions. The nearer the tropics, the greater the incidence of the disease. It is uncommon in North China but common in South China and it is endemic in West Africa. The disease is due to an ultra microscopic virus and is spread by sexual intercourse. Rectal strictures may occur in both males and females. In females the stricture is secondary to direct invasion of the rectal lymphatics from a posteriorly placed lesion on the vagina. In males, the stricture follows homosexual practices and may be preceded by a period of resistant chronic proctitis and the classical inguinal bubo. There is no doubt that many of the strictures which in the past were labelled syphilitic, actinomycotic or tuberculous were, in reality, cases of lymphogranuloma venereum.

The following tests may be made: (1) the Frei test, made with a yolk sac antigen and known as the Lygranum skin test, (2) the Complement fixation test, and (3) biopsy to exclude malignant growth. It should be pointed out that a positive Wasserman reaction does not necessarily mean that the rectal stricture is due to syphilis as the two venereal diseases may have been contracted at one and the same time.

Treatment of Fibrous Stricture due to Lymphogranuloma Inguinale. In the stage of proctitis the disease may yield to the modern powerful antibiotics such as aureomycin, terramycin, and achromycin. Hawe (1951) reports the failure of a defunctioning colostomy combined with chemotherapy. With this treatment, local sepsis persists, the infection may spread upward from the rectum to the distal colostomy loop. If the stricture does respond initially to dilatation, subsequent closure of the colostomy may be followed by recurrence. For very extensive strictures, involving the anal margin, a rectal excision and permanent colostomy is necessary. In strictures of the middle or upper third of the rectum encouraging results have been obtained by excision of the stricture and re-establishment of continuity of the bowel. The latter type of operation should be done in two or three stages owing to the difficulties encountered in removing a chronic inflammatory rectal tumour.

Following Irradiation of Carcinoma of the Cervix

A rectal stricture may follow the acute proctitis and ulceration resulting from accidental local overdosage in the course of treatment of a carcinoma of the cervix by radium. It may be due to slipping and displacement of the vaginal radium applicators or to retroversion of the uterus when the intra-uterine applicator rests against the rectum. The disease is heralded by a proctitis which is followed by a chronic ulcer on the anterior rectal wall strongly resembling a rectal carcinoma. The sigmoidoscopic appearance of the ulcer is typical, there is a minimal shelving edge with a dirty white or yellowish floor to the ulcer. Pathologically, there is an initial mucosal necrosis followed by the formation of a perirectal mass of fibrous tissue, this mass being quite out of proportion to the size of the ulcer. There is a very widespread vascular occlusion and endarteritis in the surrounding tissues and this factor makes any local operative procedure doomed to failure. A permanent colostomy is usually necessary for severe radium ulceration and necrosis of the rectum.

encountered by the author remained obscure until laparotomy. Repeated biopsies of the stricture showed chronic inflammatory tissue only, all the usual clinical tests, *including the Frei and Wasserman reaction, were negative. At laparotomy the patient,* a well-covered male of 66, was found to have a diffuse carcinoma of the stomach (linitis plastica) with secondary deposits in the liver and secondary plaques in the recto-vesical pouch which were undoubtedly responsible for the fibrous reaction in the perirectal tissues.

Non-malignant strictures of the rectum may be congenital, traumatic, or inflammatory.

(1) Congenital Stricture of the Rectum. This may occur at the anal margin or up to 4 cm. within the anal margin. The child usually presents with abdominal distension and a history of having passed ribbon stools These cases can be treated successfully by regular dilatation. Baron (1953) has reported a case of congenital diaphragmatic stricture in the rectal ampulla associated with a cord-like stenosis of the sigmoid colon and a retained left testicle.

(2) Traumatic Strictures of the Rectum may follow the accidental administration of hot enemata

They may occur as a sequel to certain operations on the anal canal and rectum. Post-hæmorrhoidectomy strictures occur at the anus and are due to the removal of too much anal mucosa and lack of post-operative supervision, the wounds in the anal canal being allowed to heal without the anus being dilated to its normal size. A rectal stricture may arise at the anastomosis of a recto-sigmoidectomy for rectal prolapse or a resection of the rectum by the abdominal route, as in anterior or abdomino-anal resections. It may also follow the diathermy fulguration of a large papilloma or the diathermy removal of multiple small polyps seen in polyposis intestini.

(3) Inflammatory Strictures of the Rectum. These may occur following *war wounds* where there is extensive destruction of the pelvic tissues and prolonged infection. They also occur from infective gangrene of the rectum as a sequel to neglected thrombosed hæmorrhoids. A rectal stricture may be secondary to a long standing horseshoe fistula whose main track lies on the under surface of the levator ani muscle and almost completely surrounds the rectum. If the fistula is laid open and drained correctly, the rectum dilated gradually and the dilatation continued during the healing period, the stricture will resolve.

An impacted foreign body lying transversely across the rectum, such as a rabbit bone or large fish bone, can also cause a rectal stricture, the diagnosis of which can be extremely puzzling.

Crohn's Disease. Occasionally a chronic inflammatory rectal stricture is encountered for which no cause can be found and which clinically and histologically strongly resembles the type of chronic lesion known as Crohn's disease It is not unreasonable to suspect that rectal strictures may be secondary to this scarring, stenosing lesion which has been described throughout the intestinal tract from the œsophagus downwards

Ulcerative Colitis. Rectal strictures occur as a late complication of ulcerative colitis and are usually associated with secondary infective lesions, such as fissure, fistula, and anal ulceration. The rectal wall becomes rigid, control of the fluid fæces becomes indifferent and the perianal skin develops an intractable soreness. When ulcerative colitis is complicated by rectal strictures the only treatment worthwhile considering is a total

habit of assisting defæcation by digital removal or who digitate the rectum for neurotic reasons.

(6) NON-SPECIFIC CHRONIC ANAL ULCER. Finally, there is a group of cases of chronic anal ulceration in whom the ulcers show no clinical characteristics and no known tests assist in the diagnosis. These cases cause a great deal of worry and anxiety not only to the patient himself but to his medical attendants, and not infrequently a prolonged period of investigation ends in a rectal excision.

ANO-RECTAL ABSCESS

The connective tissue round the anus and rectum may become infected with pyogenic organisms as a result of direct infection, local extension from a neighbouring focus of suppuration, or by the blood stream. It is usual to classify these infections into:

(1) Perianal abscess
(2) Submucous abscess
(3) Ischio-rectal abscess or
(4) Pelvi-rectal abscess

and the classification is based on the anatomical space which may be involved.

(1) *Perianal Abscess.* Infection of the perianal space may result from an infected hair follicle or sweat gland, or it may be secondary to an infected anal hæmatoma or fissure-in-ano. It may also result from spread of a submucous abscess. A very troublesome form of perianal abscess may arise following the injection of oily anæsthetic solution for the relief of pain or irritation. Such an infection can only be a direct infection due to failure of aseptic surgical technique and it cannot be too strongly emphasized that when injections of this sort are given particular care must be taken to see that infection is not introduced.

(2) *Submucous Abscess.* This is an unusual form of abscess which is generally due to infection of the submucosal space either from a sharp, ingested foreign body or injury of the mucosa introduced from below. It may also occur from an infected pile or after an injection for hæmorrhoids.

(3) *Ischio-rectal Abscess.* Infection of the ischio-rectal space may be secondary to any of the minor infected lesions of the anal canal. Infection can also spread down the vestigial anal intermuscular glands which have their opening at the level of the anal valves. It is possible that sometimes the ano-rectal glands suppurate, as a result of lymphatic infection, to form an abscess in the ischio-rectal fossa.

(4) *Pelvi-rectal Abscess.* This is an infection of the supra levator space and is secondary to intra-abdominal lesions such as an appendix abscess, pyosalpinx, Crohn's disease, tuberculous, or actinomycotic enteritis. The supra levator space may, however, be infected from below, particularly by the injudicious use of an exploring probe.

Signs and Symptoms. The symptoms of the abscess may be preceded by those of the anal lesion to which it is secondary. To the general symptoms of malaise, anorexia, and headache is added a local throbbing pain. On examination there may be no visible external signs. The sphincter will be in spasm and digital examination resented. Perianal abscesses are quite evident and present all the usual signs of local inflammation.

Treatment. An ano-rectal abscess may be drained as soon as it is diagnosed. In the

The successful treatment of a rectal stricture can only follow a correct appreciation of its ætiology. The diagnosis is based on a thorough clinical history and examination, an awareness of the behaviour of the various forms of local pathology, and the intelligent application of modern clinical laboratory methods.

References

Baron, H. W. A. (1953) *Proc. Roy. Soc. Med.* 46, 621.
Hawe, P. (1951) *Proc. Roy. Soc. Med.* 44, 426.

ANAL ULCERATION

THE commonest ulcer at the anal margin is that of a chronic fissure which is usually situated in the mid-line posteriorly or, less frequently, anteriorly. There remains a group of specific ulcers in which the differential diagnosis is by no means easy.

(1) ATYPICAL MALIGNANT ULCERS. These may be seen as erosive ulcers on the perianal skin or as small ulcers resembling a fissure except that their position may not be mid-line. These ulcers are usually transitional or squamous celled carcinomas, frequently of a high grade of malignancy. A rodent ulcer may also present round the anus. The diagnosis is established by biopsy which may have to be taken under anæsthesia.

(2) TUBERCULOUS ULCERATION. Tuberculous ulceration may occur at the anal margin or in the rectum. The margin of the ulcer has a characteristic mauve, indolent appearance without induration and the edges are typically undermined. It may be almost impossible to determine between an early carcinoma and a tuberculous ulcer, even when the specimens are in a museum jar. Radiological examination of the chest may reveal infection of the lungs. If tuberculous ulceration is suspected, the ulcer should be excised with diathermy and the floor of the ulcer coagulated. Biopsy of the inguinal glands may show a secondary infection with the presence of tubercle bacillæ.

(3) SYPHILITIC ULCERATION. A primary chancre at the anal margin is seen once or twice every year in the out-patient department at St. Mark's Hospital, and the surgeon must always be alert for this possibility. It frequently presents as an anal fissure but differs from an anal fissure in the following respects: it is larger, it is not in the usual position, and there is considerably less spasm and pain than might be expected. Examination of the inguinal region may reveal a classical bubo and a dark ground illumination of a washing from the floor of the ulcer shows the presence of spirochætes.

(4) AMŒBIC ULCERATION. Extensive, intractable ulceration may complicate amœbic dysentery, the primary infection having occurred some time in the past. If the vegetative or cystic forms of the entamœbahistolytica can be demonstrated the management of the case is obvious, but it may be necessary to give a trial therapeutic course of emetine to establish the diagnosis.

(5) SOLITARY ULCER. There is a form of ulceration which occurs about 10 cm inside the anal margin. It is a flat, erosive type of ulcer with a yellow floor and a bright red, hyperæmic margin It is frequently seen on the middle rectal valve and may vary greatly in size and shape. No one has definitely established a cause for the condition but it is worthwhile noting that the ulcer is usually well within reach of the average length index finger and may be caused by trauma, especially in patients who have a

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between the legs with a bland curd soap, but it should be seen that the facilities for this are possible.

If the irritation becomes intolerable, the patient must be instructed to pinch the skin through his underclothes, rather than abrade the superficial epithelium by scratching. If the scratching occurs while the patient is asleep at night, cotton mittens should be worn. Wool should not be worn next to the skin, but underclothes of cotton, ærtex, or celanese. In the winter a set of woollen underwear may be worn on top of the cotton.

If a local lesion exists, treatment of the local lesion, such as hæmorrhoid injection or removal, division of the internal sphincter for a fissure, and laying open of a fistula, will stop the irritation. It is interesting to note that as a wound in the anal region heals the skin begins to irritate and the irritation persists until some time after healing is complete.

The following local application may be used on the skin night and morning and during the daytime, if the opportunity exists, when the irritation is severe:

Phenol . . .	gr. 15
Zinc oxide . .	gr. 30
Prepared calamine .	gr. 15
Glycerine . .	m. 30
Spirit recti . .	m. 30
Aque rosae . .	dr. 1
Magnesii hydratus	
	to the ounce.

Chronic Resistant Cases. In these cases the assistance of a dermatologist and psychiatrist may be sought. There is no doubt that sometimes a patient confronted with an insoluble problem develops pruritus as an expression of his need to "scratch in between" the conflicting aspects of his problem.

X-rays may give temporary relief but the irritation almost invariably returns in a severe and intractable form.

Alcohol Injection. Relief can be obtained by injecting 75 per cent or absolute alcohol under the skin. It should be done with strict aseptic precautions. A grid of 1 centimetre squares should be marked by needle scratch on the perianal skin. Using a fine needle and an accurately ground 1 cc. glass syringe, 3 m. of alcohol are injected under the centre of the skin in each square It is quite useless to use a larger bore metal piston syringe as the volatile alcohol seeps backwards into the barrel above the piston and the dosage becomes uncontrolled and inaccurate.

References

Graham, J. W. and Goligher, J C. (1954) *Brit. J. Surg.* 42, 151.
Goligher, J. C; Lloyd-Davies, O. V.; Robertson, C. T. (1951) *Brit J. Surg.* 38, 467.

ischio-rectal variety it is a mistake to wait until the abscess "points," until fluctuation can be elicited, or until the perianal skin becomes red. The time-honoured application of fomentations until these signs are manifest must be strongly condemned; to wait until the abscess "points" is to wait until the infection has spread from the ischio-rectal space to the perianal space. To avoid the formation of a fistula the abscess must be drained in its early stage. A cruciate incision is made over the abscess and the roof of the abscess cavity removed. A specimen of pus should always be sent for examination and a small portion of the abscess wall should be sent for microscopic section. Supra levator abscesses should be drained secundum artem.

Crohn's disease is notorious for producing ano-rectal infections and remaining latent itself. It has already been pointed out that if the wound of an ano-rectal infection does not heal, Crohn's disease should be suspected.

Most fistulæ follow ano-rectal abscesses and it is by the early efficient drainage of these abscesses that many fistulæ can be avoided.

PRURITUS ANI

By "pruritus ani" is understood an irritation of the perianal skin and the skin lining the lowest part of the anal canal.

It may be a temporary symptom lasting a few weeks or a chronic, persisting irritation which lasts for years. It is frequently secondary to minor ano-rectal lesions.

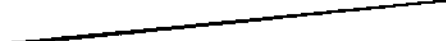
History and Examination of the Patient. The irritation is, as a rule, worse on first getting into bed at night. It may come on after, or be aggravated by, defæcation. It is more common in the summer than in the winter months. The patient should be asked about previous skin disease and any skin eruption elsewhere on the body; a history of asthma, hayfever or urticaria may point to an allergic factor. Other members of a household suffering from the same complaint may be a clue that threadworms are responsible. In chronic cases, previous treatment must be carefully detailed and inquiries made into any medicines, drugs, or local application that the patient is using.

The examination must include a general examination of the patient with special attention to other warm, moist areas of skin on the body, such as under the arms, under the breasts, in the groins, and between the toes. The urine must be tested.

Local examination is directed to excluding fissures, fistulæ, hæmorrhoids, and threadworms. The condition of the perianal skin itself may vary tremendously. Tell-tale scratch marks are usually present and may be an index of the severity of the symptoms or to the patient's lack of control. The perianal skin may be thrown into whitish, sodden, hyperkeratotic skin, in some cases closely resembling leukoplakia. A common type is a localized zone of dermatitis where the skin of the buttocks appear to have rubbed together. Occasionally, the whole perianal skin may be bright red and blistered These patients have usually used one of the anæsthetic ointments for too long a period, their skin has become sensitized to the drug and the lesion is now a "dermatitis medicamentosa."

Treatment. In the majority of cases a cure or a great improvement can be achieved with soap and water and common-sense instructions on anal hygiene. It must be remembered that in many people soap and water cleanse the perianal skin but once a week. Not only must the patient be encouraged to take a daily bath with a careful wash

INDEX



INDEX

427

MILITARY SURGICAL MANUALS

NATIONAL RESEARCH COUNCIL

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II. Ophthalmology and Otolaryngology
III. Abdominal and Genito-urinary Injuries
IV. Orthopedic Subjects
V. Burns, Shock, Wound Healing, and Vascular Injuries
VI. Neurosurgery and Thoracic Surgery

FOREWORD

THE Medical Department of the Army has been confronted with the necessity for enormous and rapid expansion paralleling that of the armed forces. The state of war has greatly increased the task of furnishing adequate medical care for Army personnel since battle casualties are added to the already wide range of diseases and injuries that must be treated.

Expansion of the medical establishment of the Army is entirely dependent on entry into the service of individuals from civil life. While most reserve officers have had a varying amount of military training, practically all medical officers will encounter problems in the military service entirely foreign to their previous experiences. These problems are by no means confined to those of an administrative nature; many are distinctly professional. The military situation imposes certain restricting factors which render impracticable some procedures that would be considered ideal in civil life. The goal of furnishing the best possible treatment to all individuals is the same in the Army as in civil life, but the means to attain that goal may differ materially.

There has been a marked tendency to specialization within the medical profession since the first World War. This tendency is fundamentally sound but does serve to increase the problems of many individual medical officers in time of war. Specialization cannot be followed to the same degree in the military service as in civil life. While many highly qualified specialists in the various fields of medicine and surgery will serve in like capacities in the Army, this cannot invariably be true. The great burden of medical care will fall on medical officers outside the highly specialized fields. It is thus essential that nearly all medical officers be familiar with the principles of military surgery. Recent advances in therapy have resulted in radical modification of certain principles of treatment that were formerly considered sound.

This series of texts presents in compact form essential up-to-date and reliable information regarding military surgery. The various sections have been written by outstanding authorities in their respective fields. They have been prepared for publication under the auspices of the Division of Medical Sciences of the National Research Council.